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PANCREATIC CARCINOMA AND ITS EARLY ROENTGENOLOGIC RECOGNITION*

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THE clinical diagnosis of malignant tumors of the pancreatico-ampullary area is often difficult and, because of their rapid growth, treatment in late stages quite unsatisfactory. Within the last several years a number of papers reported long term cures due to advances in operative techniques and the proved feasibility of radical pancreatico-duodenectomy. These long term results refer to neoplasms located in the head of the pancreas and adjacent duodeno-ampullary area since carcinomas of the body and/or tail either produce early metastases or are usually too advanced to be amenable to a successful operation. Therefore, an early recognition of pancreatico-ampullary neoplasms can prove to be life-prolonging, particularly so in the clinically nonjaundiced patient. These tumors can often be diagnosed by roentgenologic means on routine upper gastrointestinal studies when other clinical characteristics are still too indistinct, and the radiologist can thus play an important role in their early recognition. According to various reports, a correct preoperative roentgenologic diagnosis is made in about 30 per cent to 50 per cent of cases, including both the jaundiced and the nonjaundiced patients. In the jaundiced patient, there is—to a great extent—a "built-in" diagnosis, and usually in these cases the roentgenologic changes are obvious. With increased alertness and attention to the subtle, early roentgen signs, a correct preoperative diagnosis could perhaps be made in about 75 to 85 per cent of the cases, excluding tumors of the body and tail. In the latter location, it is difficult, if not impossible, by routine examination to detect the early roentgenologic signs of neoplasms. It should be emphasized that, although the roentgen manifestations might appear to be minor, the tumor may be found to be inoperable due to local extension or metastases. The location of the carcinoma is more important than its size, and small tumors arising near the duodenal wall may produce early roentgen changes as well as compression of the common duct with accompanying jaundice.

Excellent general reviews discussing the frequency, diagnostic criteria and the symptomatology of pancreatic neoplasms

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were published by Berk,[2] Cliffton,[7] Bank,[1] Johnson and Kalser[15] and Gullick.[13] The roentgenologic findings were described in great detail by Case,[5] Rendich *et al.*,[26] Kline and Culver,[16] Poppel,[24] Hodes *et al.*,[14] and others. In this paper, an attempt is made to emphasize the need for careful examination and familiarity with early, rather subtle, roentgenologic changes which could increase the preoperative diagnostic accuracy.

INCIDENCE AND LOCATION

According to statistics, carcinoma of the pancreas accounts for 1 to 4 per cent of all cancers, and about 17 per cent of cancers arising in the stomach, liver, biliary ducts and pancreas (Frantz[10]). It is said that carcinoma of the stomach occurs about four times as frequently as in the pancreas, but in our series this was not the case. During the years 1950 through 1959, 91 cases of carcinoma of the pancreas and 224 cases of carcinoma of the stomach were seen in our institution, corresponding to a ratio of 2.5:1. These neoplasms are more common in the male, the ratio being 2:1. They may occur in the younger age group, but the highest incidence is found between the ages of fifty to seventy. Carcinoma occurs in the head of the pancreas in about 65 to 80 per cent of cases; it is far less common in the body and tail, but sometimes it is difficult to establish the origin of the neoplasm as it might extend from the head into the body or involve the head and/or body and/or tail. Carcinoma is observed three times more frequently in the pancreas than in the papilla of Vater or the common duct. Tumors of the pancreaticoampullary area can arise from any of the several structures which are located in close proximity: the head of the pancreas, the common bile duct, the ampulla and papilla of Vater, the duct of Wirsung and/or Santorini, or the duodenal mucosa. A neoplasm arising in this area may envelop the surrounding structures to the extent that its exact origin is often obscured. As the long term cure rate of carcinoma of the ampulla of Vater and duodenum is much more satisfactory than that for carcinoma of the head, it is important that a preoperative or operative distinction be made.

LABORATORY DATA

According to Cattell *et al.*,[6] the majority of patients have elevated bilirubin values, ranging from 1.3 to 38 mg. per 100 cc.; in 50 per cent of their cases the serum bilirubin was below 10 mg. per 100 cc. and in 85 per cent below 20 mg. They stated that one should be hesitant with the diagnosis of carcinoma when the bilirubin value is less than 3 mg. or greater than 35 mg. We found that in the nonjaundiced patients the values were lower than 3 mg., but that, roentgenologically, changes were discernible which were compatible with the presence of a malignancy in the pancreatico-ampullary area. Alkaline phosphatase and cholesterol values are elevated, depending on the type of obstructive jaundice. In the beginning the values in flocculation tests are insignificant, but with recurrent cholangitis changes in flocculation values occur. Serum and urinary lipase and amylase may be elevated in patients when the pancreatic duct is blocked, but the values may be low if the obstruction has been prolonged with subsequent destruction of the secreting pancreas. Also, an investigation of the duodenal contents for pancreatic enzymes, sodium bicarbonate, occult blood and for cytologic changes may be of value.

PATHOLOGY AND SURGERY

A malignant tumor in the pancreaticoampullary area is usually a carcinoma and is rarely due to metastases, sarcoma, plasmocytoma, lymphosarcoma or Hodgkin's disease. Also, a malignant islet cell neoplasm (nesidioblastoma) or cystadenocarcinoma may be found. Benign tumors are usually adenomas or cystadenomas, dermoid cysts, composite tumors, nonepithelial benign tumors, or benign islet cell tumors (Frantz[10]).

Fraser[11] reviewed 1,035 cases of obstruc-

tive jaundice caused by pancreatic lesions and found that 74 per cent were due to neoplasms; 7 per cent were diagnosed as malignant although only an inflammatory disease was present, and in 16 per cent of cases pancreatitis was diagnosed when a neoplasm was present. Bowden[4] has pointed out that at times, at the operating table, it is often difficult to differentiate cancer from chronic pancreatitis, and that one may encounter a disparity between the size of the gross tumefaction in the head and that of the neoplasm which may be very small but seems larger because it is sur rounded by a large inflammatory component. Biopsy is often avoided because of danger of pancreatitis, leakage, abscesses with fistula formation or peritonitis. Waugh and Gilbertson[29] recorded the results of operations on 85 patients up to January 1, 1953. Forty-nine traced patients survived three years or more, the rate being 28.6 per cent. Thirty-seven survived five years or more, the rate being 27 per cent. Nineteen patients were operated upon for carcinoma of the head and 3 lived five years or more, the survival rate being 15.8 per cent. Thirteen patients were operated upon for carcinoma of the ampulla and 5 lived five years, a rate of 38.4 per cent. Dennis and Varco[8] reported 2 patients with carcinoma of the head who survived six and nine years, and 3 with carcinoma of the ampulla who survived twelve, ten and nine years. Porter[25] reported 120 cases operated on between 1948 and 1952 and 130 between 1953 and 1957. In the first group 6 survived five years and 10 in the second. His patient's in general had carcinoma of the ampulla or duodenum. Millbourn[20] in Sweden reported his experience with 15 patients and found that the average survival rate in carcinoma of the head of the pancreas was twenty-three months as of September, 1958. Cattell *et al.*[6] recorded 148 patients who survived pancreatico-duodenal resection prior to February, 1958, and found that 1 out of 13 patients operated upon for carcinoma of the common bile duct was well. Fifty-five patients were operated upon for carcinoma of the ampulla of Vater, and of these 12 (36.8 per cent) lived five or more years; 13 patients were operated upon for carcinoma of the duodenum with 3 (37.5 per cent) surviving five years. Sixty-three patients were operated on for carcinoma of the head of the pancreas and of these 4 (9 per cent) survived for five years. Zintel *et al.*[31] reported 83 patients operated on for carcinoma of the pancreatico-ampullary area; 6 of these survived five to eleven years. None of the survivors was of the group in which the process began as carcinoma of the pancreatic acini.

SYMPTOMATOLOGY

The clinical signs and symptoms can be divided into early and immediate preoperative.

I. *Early Symptoms:*

A. Contrary to the widely held belief that painless jaundice is an early characteristic symptom, one of the earliest and most common complaints is pain which often precedes jaundice, and, in involvement of the body and tail, it may constitute the only symptom. The pain is usually not diagnostic, but three main types may be encountered: (a) a persistent, dull mid-epigastric pain radiating to the lower back, (b) pain, colicky in nature, usually in the right upper quadrant, extending to the subscapular area, and (c) a paroxysmal pain in the mid-abdomen radiating to the anterior part of the chest and back. Sometimes these patients visit doctor after doctor, and are labelled as psychoneurotics. Occasionally, they develop emotional disturbances, characterized by depression and anxiety, which can probably be traced to a fruitless search for medical help. The nature of the pain, its extent and location depend on the site of the tumor and the presence of local or distant metastases. In a series of 88 patients without jaundice, it was found that when the neoplasm involved the body and/or tail, the pain was generally on the left and in the back, while in tumors of the head the pain was usually on the right. In

carcinoma of the head, the pain is influenced by the activity of the upper gastrointestinal tract and is aggravated by intake of food, with relief following passage of stool or flatus. In involvement of the body and/or tail and, to a lesser extent, the head, relief from the pain can be obtained by a change in posture, as the pain seems to be more intense in horizontal position than in the upright or decubitus positions.

B. Another consistent symptom is weight loss. In carcinoma of the head, weight loss is more marked than when the papilla of Vater is initially involved, for in the latter jaundice occurs at an earlier stage. Weakness, anorexia, nausea, flatulence and vomiting are found in these patients.

C. Patients may complain of increasing constipation (which, in the older age group, is difficult to evaluate) and also of sudden onset of unexplained diarrhea with bulky stools primarily due to undigested fat and protein. This unexplained steatorrhea can be found in about 22.2 per cent of cases.

D. Gastrointestinal bleeding is noted in 25 to 37 per cent of cases, mainly in lesions of the duodenum and of the ampulla of Vater. However, it can occur in carcinomas of the head when, apparently, the tumor erodes and bleeds into the duct of Wirsung and thence into the duodenum.

E. Migrating thrombophlebitis, in our experience, was not a common occurrence, although the literature is replete with reports in this respect.

F. Pancreatic carcinomas occur more often in diabetics, but an abnormal glucose tolerance curve is frequently found in the nondiabetic patient.

II. *Immediate Preoperative Signs and Symptoms:*

A. Jaundice, usually progressive. Berk,[2] in his series of 1,361 cases, found that jaundice occurred in 64.7 per cent at some time. In carcinomas arising in the head, 81.3 per cent had jaundice, and in papillary neoplasms jaundice was present in all of the patients. Sometimes jaundice regresses temporarily due to necrosis and sloughing of a polypoid tumor about the papilla of Vater. Generally, in carcinomas of the head pain precedes jaundice; in ampullary malignancy jaundice might precede pain. Painless jaundice is relatively rare, and has been reported to occur in 8.8 to 26.5 per cent of cases in different series.

B. Pain, similar to that described in the early stage, but more relentless and severe.

C. Rapid weight loss with anorexia, marked weakness and pronounced gastrointestinal disturbances.

D. A palpable gallbladder is present in about 27 per cent of cases; enlargement of the common duct is more frequently present than the Courvoisier gallbladder (this applies more aptly to the common duct).

E. Enlarged liver (Schiff[27]).

F. Chills and fever.

ROENTGEN EXAMINATION

Over the past decade we have been interested in the possibility of improving the accuracy in diagnosis of pancreatic carcinomas by routine upper gastrointestinal examinations. Between January, 1950, and December, 1959, 91 cases of pancreatic carcinoma were operated upon or autopsied in our institution. Sixty-four of these were examined in our department and 49 recognized preoperatively; 47 were in the head, 10 in the head and body, 4 in the body, 2 in the tail and 1 in the entire pancreas with a large tumefaction of the tail. The last 6 cases were not clinically jaundiced. Eighteen of the carcinoma of the head cases were also not clinically jaundiced when examined, and 6 of these were reported as roentgenologically negative. Therefore, in our entire series we have had a 77 per cent diagnostic accuracy, and in the nonjaundiced neoplasms of the head a 68 per cent accuracy. The danger of false positive cases seems to be minimal, and we are still more concerned with under-reading rather than with over-reading. In our series, only 1 case was diagnosed as a questionable small tumor in the head of the pancreas producing a pressure below the

upper duodenal knee, and at surgery no evidence of any pathology was noted. In 2 instances, the tumor was found to represent pancreatitis and not a malignancy.

Because of its anatomic location (Fig. 1), a careful conventional upper gastrointestinal series may disclose pathologic changes in the head. In our experience, spot roentgenograms of the duodenal sweep in the erect position are not too valuable because the filling of the loop is inconstant and often not adequate for diagnostic purposes. The most useful views are serialographs—with or without the use of a pressure balloon—of the duodenum completely filled with barium and taken in the prone right anterior oblique position and prone horizontal position. Sometimes, the supine left posterior oblique position proves more useful (Wise and Johnston[30]). This is due to the fact that in the latter view the duodenal loop rests on the pancreas, and nodularities of the head may produce an imprint

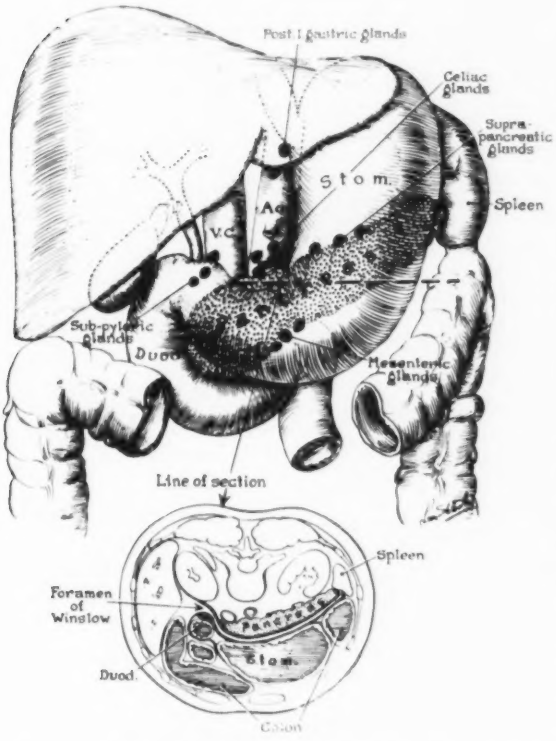


Fig. 1. Drawing showing the pancreas and its relation to surrounding structures.

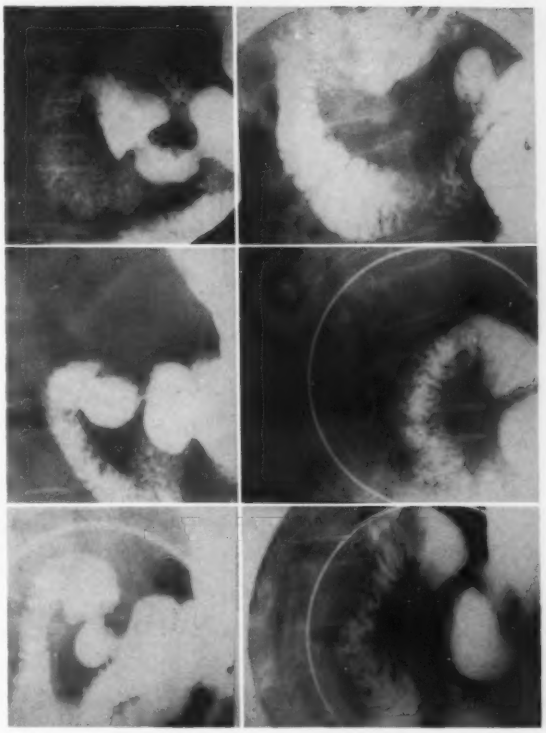


Fig. 2. Roentgenograms showing normal duodenal loop with fine feathery or slightly coarser mucosal folds.

on the duodenum, whereas in the prone right anterior oblique position the duodenum tends to fall away from the pancreas. However, it may be difficult to obtain a satisfactory filling of the duodenal loop in the supine left posterior oblique position, and the prone views are usually quite adequate.

The following early roentgenologic findings are of significance:

1. Irregular contractions, occasionally marked spasticity of some segments of the duodenum. During fluoroscopy and on roentgenograms, a retroperistalsis and disordered motor function, especially of the mesial aspect of the second or third parts of the duodenum, are noted. The normal filling of the valvulae conniventes (Fig. 2) is present on the lateral aspect of the loop but altered or absent in some mesial segments of the duodenal sweep.

2. The mucosal folds in these segments of the inner aspect of the duodenal loop dis-

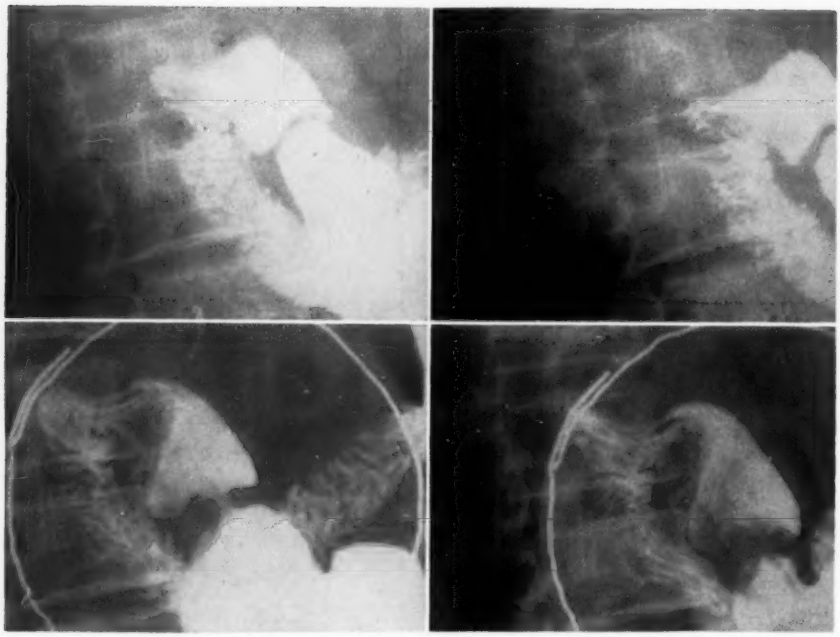


Fig. 3. B.G., a sixty-five year old female, presented a history of jaundice of three weeks' duration and an 8 pound weight loss. She had no pain. Surgery revealed carcinoma of the head of the pancreas with metastatic lymph nodes and extension into the mesentery of the ileum. Roentgenograms show the brush-like folds on the lower inner duodenal aspect and the loss of plasticity.

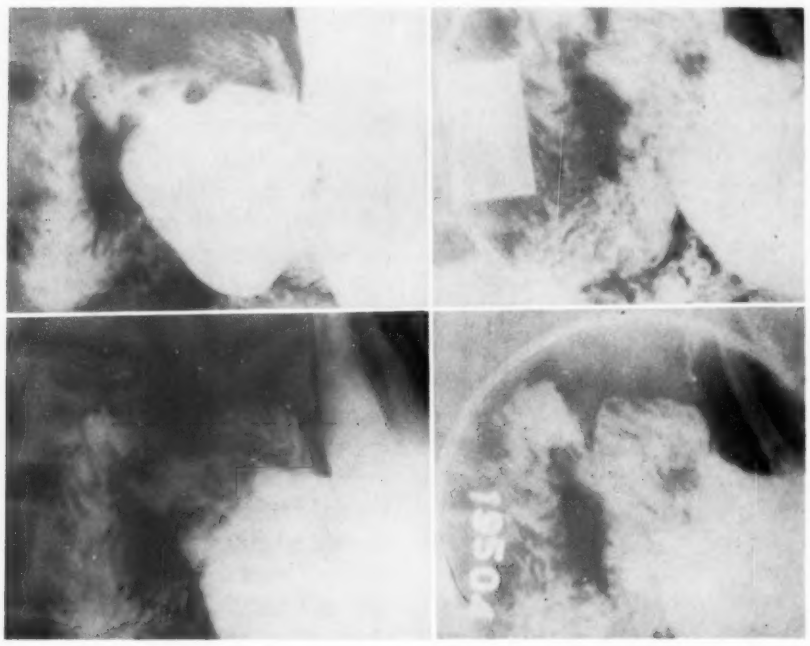


Fig. 4. S.J.M., a forty-five year old male, presented a history of several episodes of pain and nausea during the past forty-six weeks, weight loss, and jaundice of two weeks' duration. Roentgenograms show the two brush-like folds fixed in position at the proximal aspect of the medial second duodenum with flattening of the folds along this area. Autopsy revealed carcinoma of the head of the pancreas with carcinomatosis.

close changes in shape, width, height and direction. Their normal feathery appearance is absent, they assume a brush-like or spike-like configuration (Fig. 3; 4; and 5, *A* and *B*) and occasionally they are flattened and widened (Fig. 6). Some of these folds point constantly into divergent directions, as if fixed in this position by inflammatory or neoplastic changes (Fig. 7, *A* and *B*). Sometimes, the filling of the duodenum is rather fleeting in a thread-like fashion, and an irregular skeletonized ap-

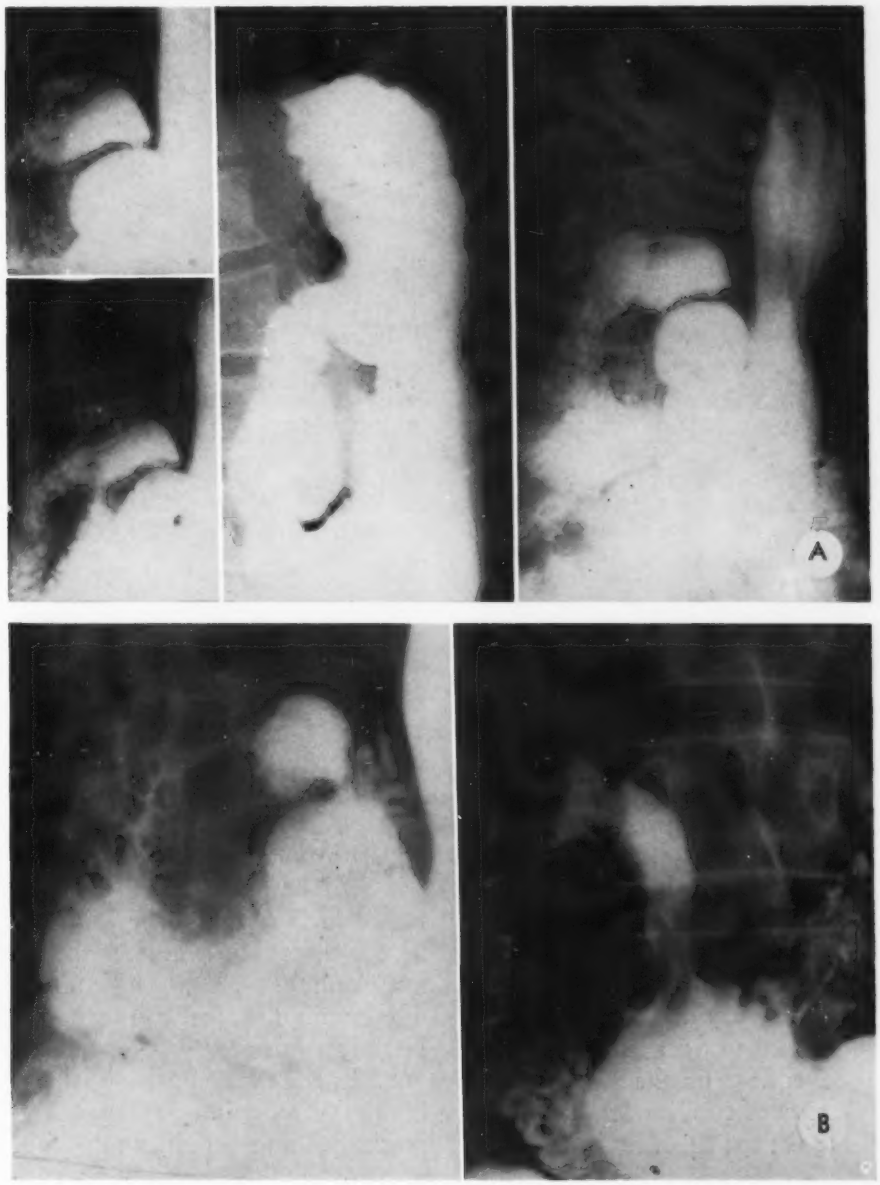


Fig. 5. T.S., a forty-eight year old female, presented a history of abdominal pain, nausea and vomiting of seven months' duration. (*A*) Roentgenograms made at our institution on May 29, 1958 were reported as negative. (*B*) Roentgenograms made in another hospital on June 12, 1959 were also reported as negative. The patient subsequently developed jaundice. Surgery revealed an inoperable carcinoma of the head of the pancreas. Note the triangular fixed fold at the junction of the second and third parts of the duodenum in *A*. Note the double contour of the duodenal loop and the shallow defects on the inner aspect in *B*.

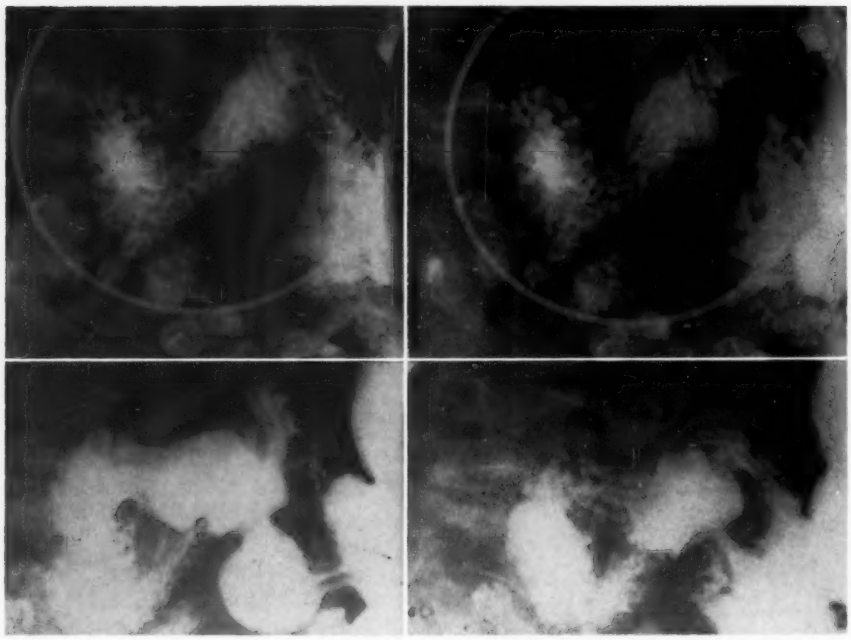


Fig. 6. R.S., a sixty-five year old female, presented a history of pain of five months' duration, which increased in severity on lying down or bending. A mass was felt to the right of the midepigastric region. No jaundice was present and liver function studies were negative. Surgery revealed an adenocarcinoma of the head of the pancreas with metastases to lymph nodes. Roentgenograms show the fixation and flattening of the folds in the lower half of the inner aspect of the second part of the duodenum. This case was not diagnosed roentgenologically.

pearance of thread-like mucosal folds is noted (Fig. 8).

3. Individual folds may become markedly elongated, presenting a pseudodiverticulum of fistula-type formation (Fig. 9). This is probably due to fixation and later necrosis with ulceration produced by the tumor.

4. Duodenal diverticula may be displaced or compressed by the mass (Fig. 10). If a large diverticulum or several diverticula are present, an investigation of the duodenal sweep is very difficult.

5. In the presence of nodularities of the pancreas, indentations on the mesial border of some segments of the duodenal loop develop (Fig. 11; 12; 13; 14, *A* and *B*; and 15, *A* and *B*).

6. With increase in the size of the mass, a concave pressure defect or a double contour of the inner half of the duodenal sweep is noticeable, especially when this area is compressed by a balloon (Fig. 16). The widened common duct produces a flattening of the inner part of the duodenum immediately overlying it (Fig. 17). These double contours are also seen when pathology other than a pancreatico-ampullary carcinoma is present.

7. Post-bulbar impression caused by a widened common duct (Fig. 18 and 19) might occur, and especially in nonjaundiced cases intravenous cholangiography may be helpful. Also, the lateral wall of the duodenum may be compressed by an enlarged gallbladder.

8. In the prone position, the second part of the duodenum is more rigid, drawn mesially, and overlies the bulb and/or antrum, as it is probably attached to the tumor (Fig. 20).

9. Carcinoma of the papilla or ampulla of Vater is sometimes indistinguishable from one arising in the adjacent structures.

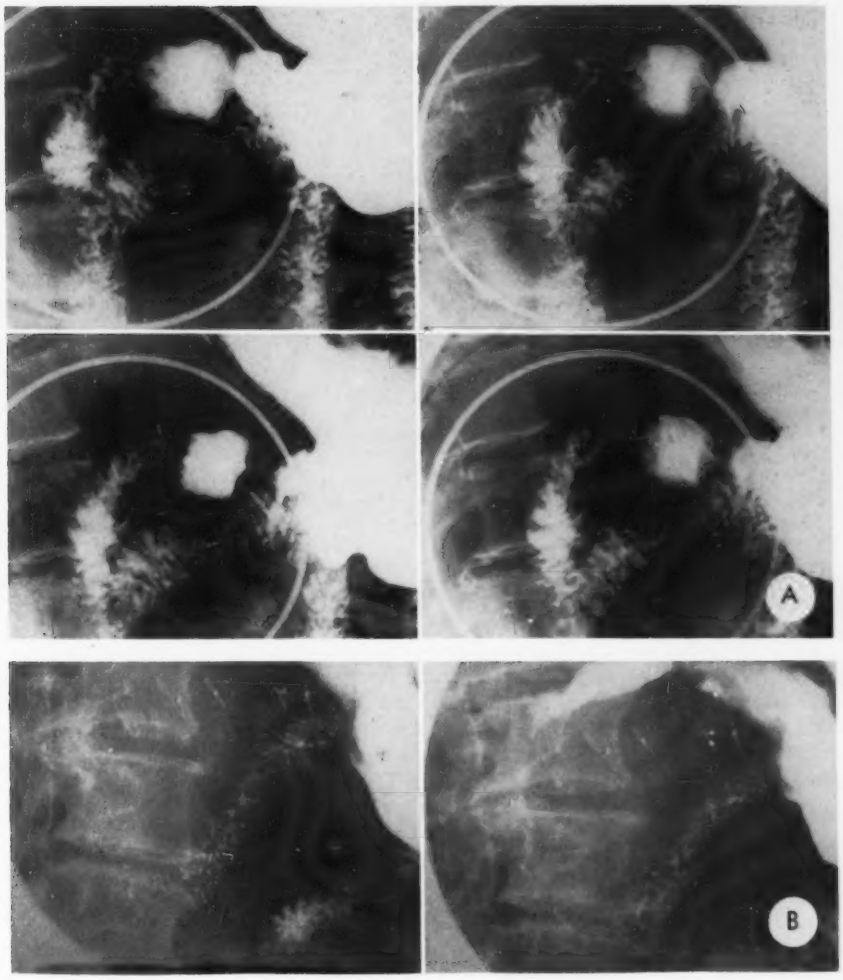


Fig. 7. M.B., a sixty year old female, presented a history of abdominal pain of seven months' duration. There was no jaundice and liver function tests were negative except for slightly high alkaline phosphatase values. Surgery revealed anaplastic carcinoma of the head of the pancreas. (*A*) Roentgenograms made April 5, 1957 show the large spike-like fold with loss of plasticity and the brush-like appearance of the mesial folds of the lower part of the second duodenum. The shallow indentation above the fold is probably caused by the tumor. (*B*) Roentgenograms made November 11, 1957 show the rapid growth of the tumor.

The mucosa is invaded and an irregular filling defect is present (Fig. 21) or an intraluminal mass is noticeable (Fig. 22).

10. With increase in the size of the tumor, the usually sought for and obvious roentgen findings appear; namely, the inverted "3" sign (Fig. 23) (Frostberg[12]), which can also be present in pancreatitis, pad (pressure) effect on the duodenum and antrum with, occasionally, considerable enlargement of the entire loop. Anterior displacement of the duodenal bulb, antrum or body of the stomach is best seen in the right lateral horizontal and left lateral erect positions (Sheinmel and Mednick[28]), and these views are most useful in involvement of the body and/or tail. These tumors of the body and/or tail often produce a change in configuration and depression of the duodenojejunal angle. Newer diagnostic methods like splenoportography and pneumotomography (Moseley[21]), in our

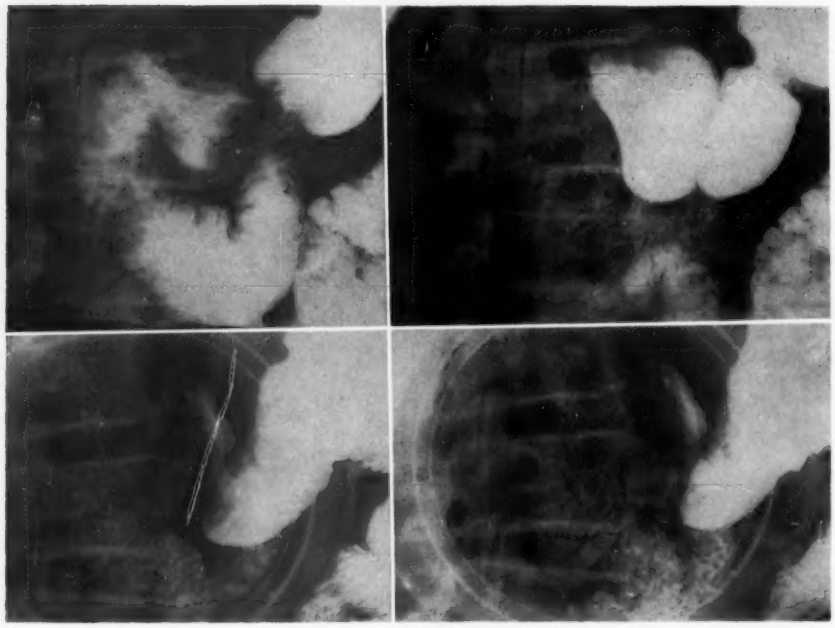


FIG. 8. B.W., a sixty year old male, had had a cholecystostomy for acute cholecystitis with cholelithiasis four months previously. There was no jaundice; alkaline phosphatase was 4.3. Surgery revealed an adenocarcinoma of the head of the pancreas. Roentgenograms show loss of normal configuration of folds at the mesial lower half of the second and third parts of the duodenum. The folds are irritable, flattened in the third part and point in different directions. On most of the roentgenograms a scanty filling of the duodenum with a skeletonized appearance of the folds is present. This case was not reported as a pancreatic carcinoma.

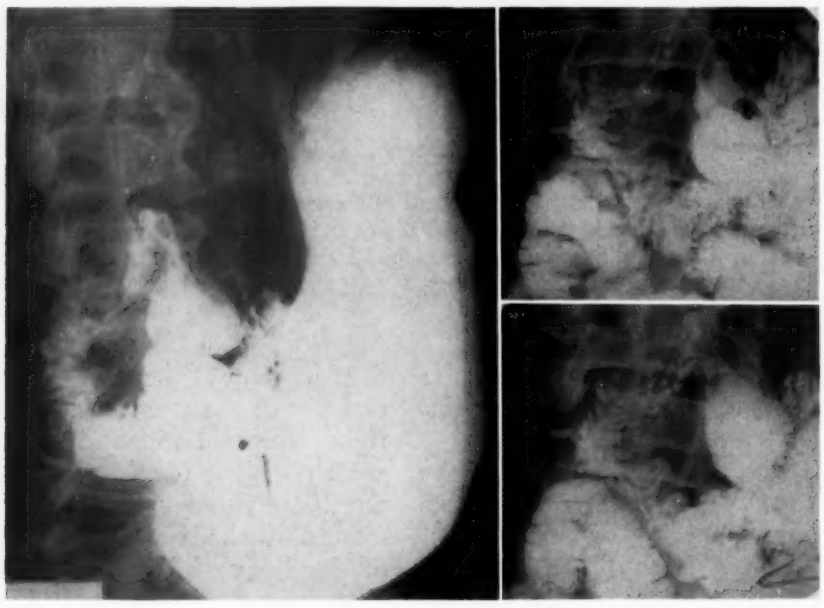


FIG. 9. S.A., a seventy year old male, presented a history of jaundice of six weeks' duration, increasing constipation, an 8 pound weight loss and continuous left upper quadrant pain for the past several months. Surgery revealed carcinoma of the head and body of the pancreas with liver metastases. Roentgenograms show an irregular spike-like fold fixed in position at the junction of the second and third parts of the duodenum with loss of plasticity, irregularity and some flattening of the folds of the third part of the duodenum and pressure by a small mass above.

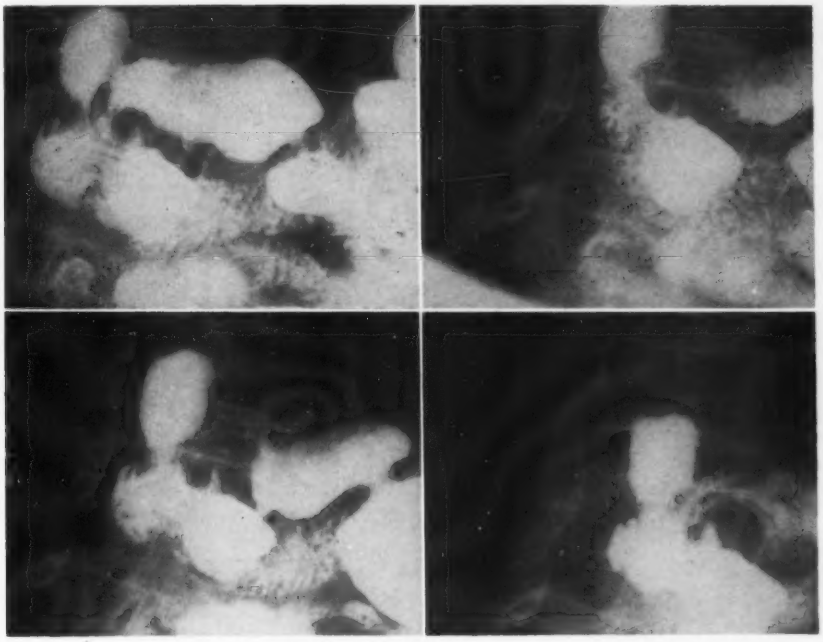


Fig. 10. J.F., a seventy year old female, presented a history of epigastric pain, anorexia and nausea of two months' duration, a 34 pound weight loss since the onset of pain and increasing constipation. She was not jaundiced. An irregular mass was felt in the right upper quadrant. Surgery revealed a lemon-sized carcinoma of the head of the pancreas. Roentgenograms show numerous diverticula, displaced laterally and anteriorly and a triangular fold fixed in position at the junction of the second and third parts of the duodenum. The upper part of the duodenal sweep appears to be compressed by a mass.

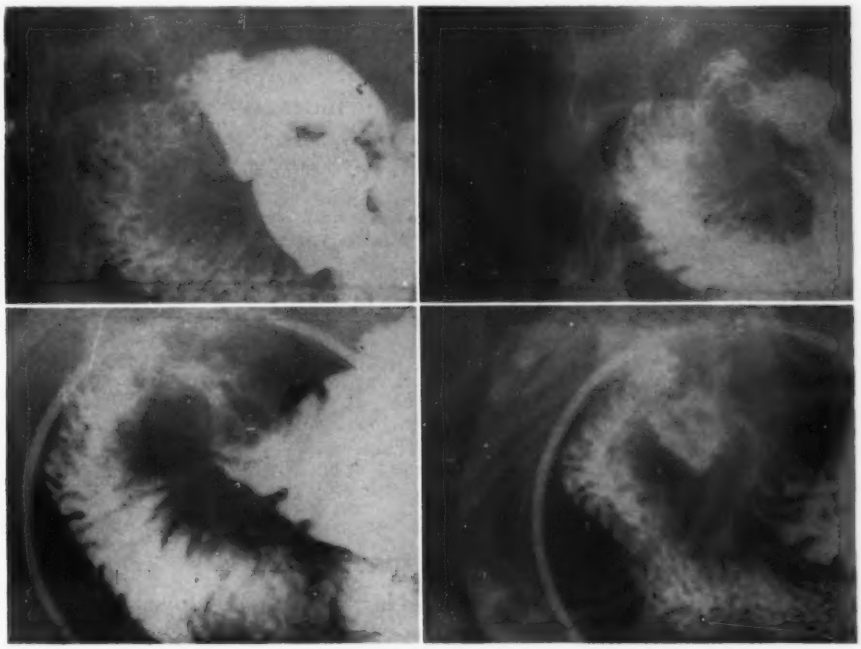


Fig. 11. McC.H., a fifty year old male, presented a history of severe right upper quadrant colicky pain with some weight loss of one and one-half months' duration. Subsequently, he developed jaundice. Surgery revealed carcinoma of the head of the pancreas. Roentgenograms show the shallow pressure defect caused by a mass and the change in configuration of the folds. A shallow indentation in the post-bulbar area due to an enlarged common duct is also visualized.

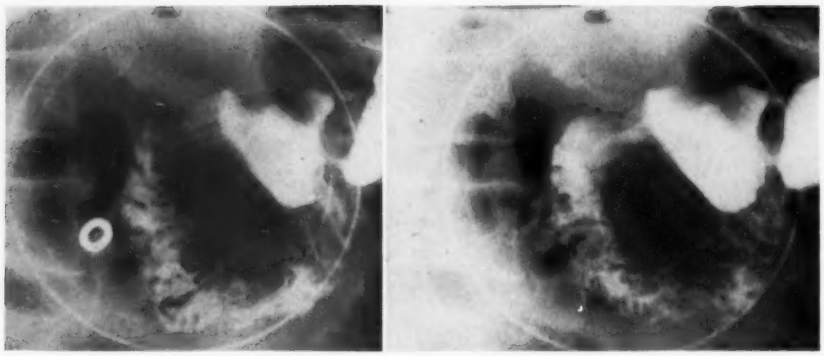


Fig. 12. S.I., an eighty-nine year old male, presented a history of a 12 pound weight loss, anorexia and fullness. Six weeks previously the patient developed jaundice which later subsided and then reappeared. Surgery revealed carcinoma of the head of the pancreas with a markedly enlarged head (10×7 cm. in diameter). Roentgenograms show irregularities of the folds at the inner aspect of the junction of the second and third parts of the duodenum with a change of direction and shallow filling defects.

opinion, disclose tumors in this category when the disease is appreciably advanced. Whenever there is a solitary or concomitant involvement of the head, and in a vast majority this is the case, a routine examination will disclose changes on the inner aspect of the loop without need for more time consuming diagnostic procedures (Fig. 24 and 25). The various stages in development of pancreatic carcinomas and their roentgenologic manifestations are shown on the drawings in Figure 26.

DIFFERENTIAL DIAGNOSIS

In the differential diagnosis, other pathologic processes occurring in the head of the pancreas, duodenum, extrahepatic biliary tree and gallbladder or adjacent lymph nodes should be considered.

I. *Pancreas*. A. The most frequent diagnostic problem is caused by pancreatitis which might produce a diffuse enlargement of the head, with compression of the inner second and sometimes third parts (Fig. 27), or localized nodularities with corresponding pressure defects of the duodenum (Fig. 28). Usually, these defects are smooth and the mucosal pattern does not disclose changes noted in pancreatic carcinomas (Fig. 29, *A–C*). Pancreatitis may compress

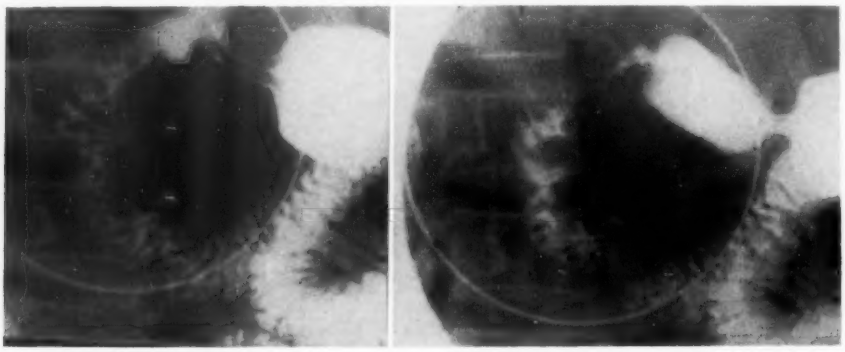


Fig. 13. D.A., a sixty-nine year old diabetic, presented a history of a sudden onset of diarrhea and jaundice of two weeks' duration. During the previous two years, the patient had three episodes of melena, but no pain. Surgery revealed carcinoma of the head of the pancreas. Roentgenograms show the pseudodiverticulum (?) and irregularities of the inner aspect of the second and third parts of the duodenum with pressure defects and fixation of folds.

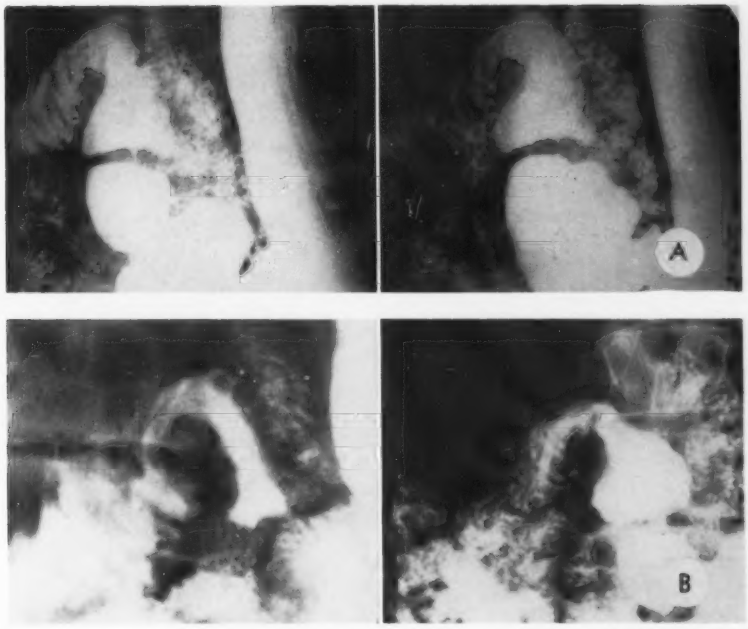


Fig. 14. H.B., a fifty-seven year old female, presented a history of abdominal cramps and nausea of a few years' duration, which three months previously increased in severity and were accompanied by a feeling of weakness, fatigue and subsequently jaundice. Surgery revealed carcinoma, the size of a small plum, located near the upper knee of the duodenum. The pancreas was removed. The patient died two and one-half years later because of metastases. (*A*) Gastrointestinal roentgenograms made October 27, 1953 were negative. (*B*) Gastrointestinal roentgenograms made August 26, 1957 show a mass below the upper knee of the duodenum. A striking difference between *A* and *B* is evident upon comparison.

the common duct and cause jaundice; an enlarged gallbladder may press on the lateral border of the duodenum, a finding which is also noted in the presence of carcinomas of the head of the pancreas (Fig. 30). Occasionally, it is impossible to make a roentgenologic distinction, and the clinical history, signs and symptoms should then be the deciding factors. A swelling of the papilla which is seen in relapsing pancreatitis (papillary sign—Poppel[23]) and possible subsequent development of pressure defects should not be mistaken for the presence of a malignancy.

B. The head of the pancreas may be the site of rare malignant neoplasms, metastases, various cysts (Fig. 31) or benign tumors. If these lesions are small enough to produce early changes on the inner aspect of the duodenal sweep, a differentiation from carcinoma manifested by similar changes might be difficult. Usually benign masses produce smooth defects without any involvement of the mucosa (Fig. 32), whereas metastases produce a roentgenologic pattern which is impossible to distinguish from one caused by a carcinoma of the head of the pancreas (Fig. 33).

C. An annular pancreas produces a quite characteristic pattern of obstruction.

II. *Duodenum.* A. Malignant lesions of the duodenum are usually adenocarcinomas of the scirrhous or papillary type. The scirrhous lesion infiltrates, destroys the mucosa with subsequent rigidity of the wall, shelf-like defects or constriction of the lumen (Fig. 34), and the papillary tumors are intraluminal producing filling defects. Involvement of the duodenum by lymphosarcoma or Hodgkin's disease (Fig. 35) distorts the mucosal pattern, produces a diffuse irregular narrowing of its lumen or a

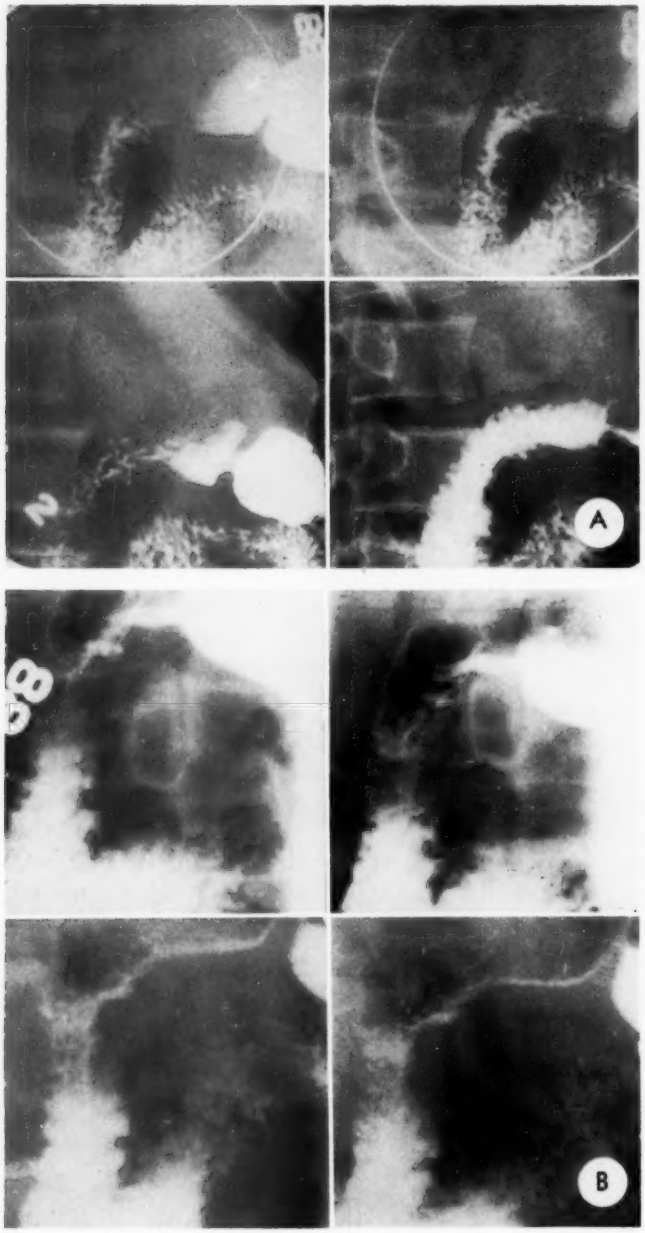


Fig. 15. W.A., a fifty-eight year old male, presented a history of pain in the abdomen, chest and increasing constipation of four months' duration. There was no jaundice. Surgery revealed carcinoma of the head of the pancreas. (*A*) Roentgenograms show the pressure defect caused by a mass in the post-bulbar area, with the folds flattened, fixed and invaded. This type of pressure defect, without involvement of the folds, is also seen in cases of pancreatitis. There is also slight irregularity and fixation of the folds of the distal third part of the duodenum. (*B*) Spot roentgenograms in the erect position. Although the findings may be discernible on the lower roentgenograms taken in a more oblique position, they cannot be visualized on the upper roentgenograms.

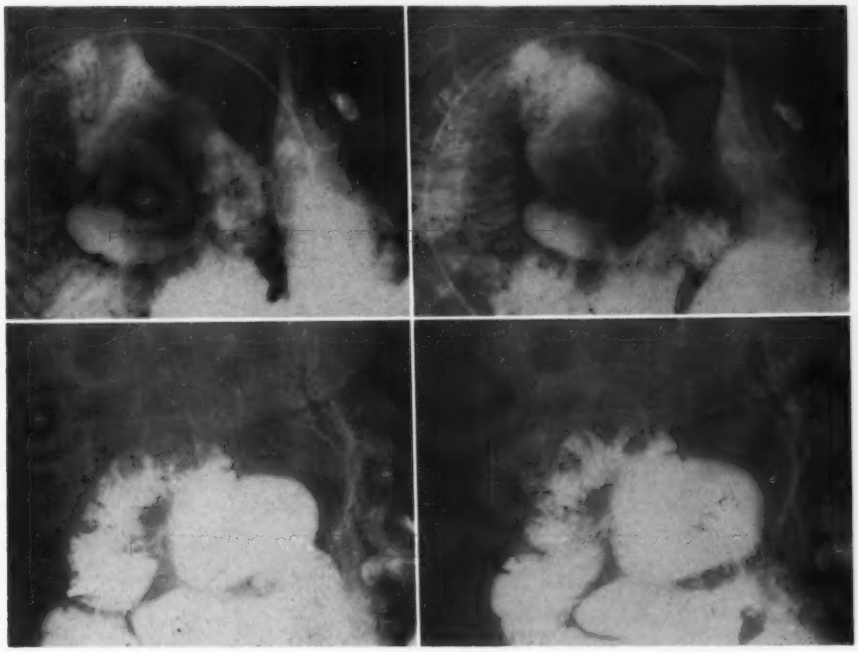


Fig. 16. H.E., an eighty-eight year old female, presented a history of jaundice of two weeks' duration, extensive weight loss and loss of appetite of two months' duration. Surgery revealed carcinoma of the head of the pancreas. Roentgenograms show the triangular fold projecting mesially and upwards from the lower second duodenal segment and the double contour on pressure studies. Also demonstrated is the post-bulbar pencil-shaped indentation caused by an enlarged common duct and diverticulum with its wall facing the elongated fold slightly compressed.

bag-like appearance when the muscle coat has been replaced by the malignant growth.

B. Post-bulbar ulcers occur probably more often than is realized, and sometimes they are responsible for secondary pancreatitis. A spastic incisura on the wall opposite the ulcer crater is usually present, and this segment of the duodenum is spastic and irritable with coarse mucosal folds. If the ulcer crater is visible, then the diagnostic problem is simplified, but occasionally only an area of irritability and spasticity of the mesial and also lateral

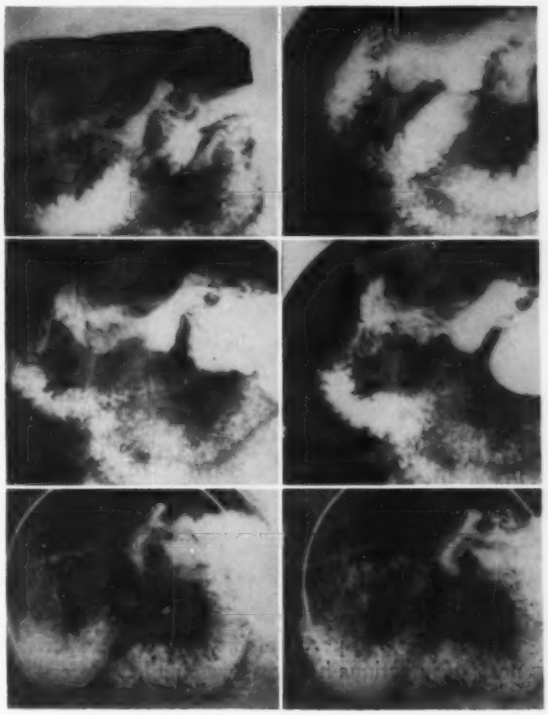


Fig. 17. K.N., a sixty-three year old female, presented a history of nausea of three weeks' duration and jaundice of one week's duration. Surgery revealed carcinoma of the common duct. Roentgenograms show the double contour of the upper inner duodenum with irregular folds.

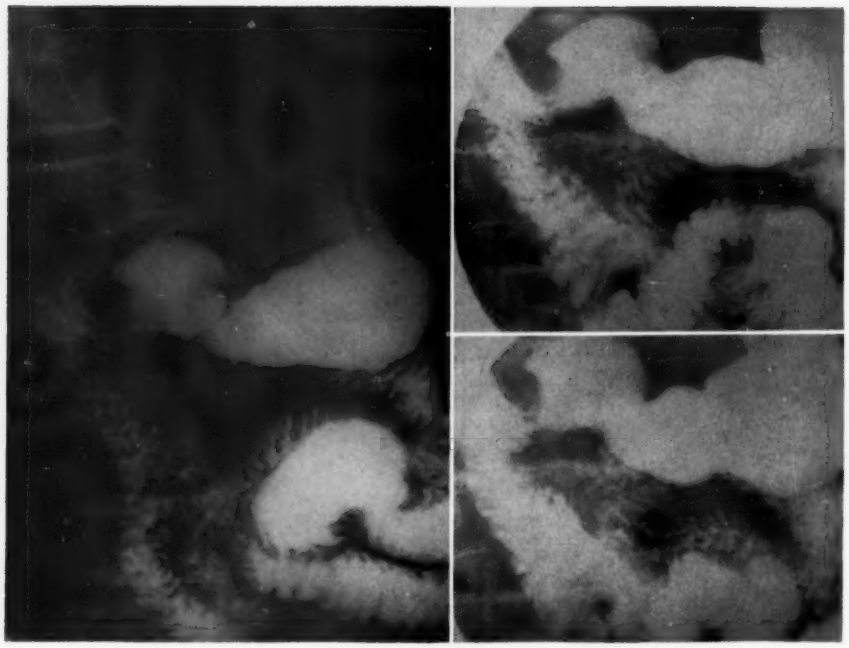


Fig. 18. B.P., an eighty-year old male, presented a history of jaundice of five months' duration. He had no pain but grew progressively weaker and anorexic. Surgery revealed a carcinoma, about 2 cm. in size, of the head of the pancreas or ampulla of Vater. Roentgenograms show the shallow pressure defect and fixation of the folds in the papillary area, also the deep post-bulbar indentation produced by a very large common duct.

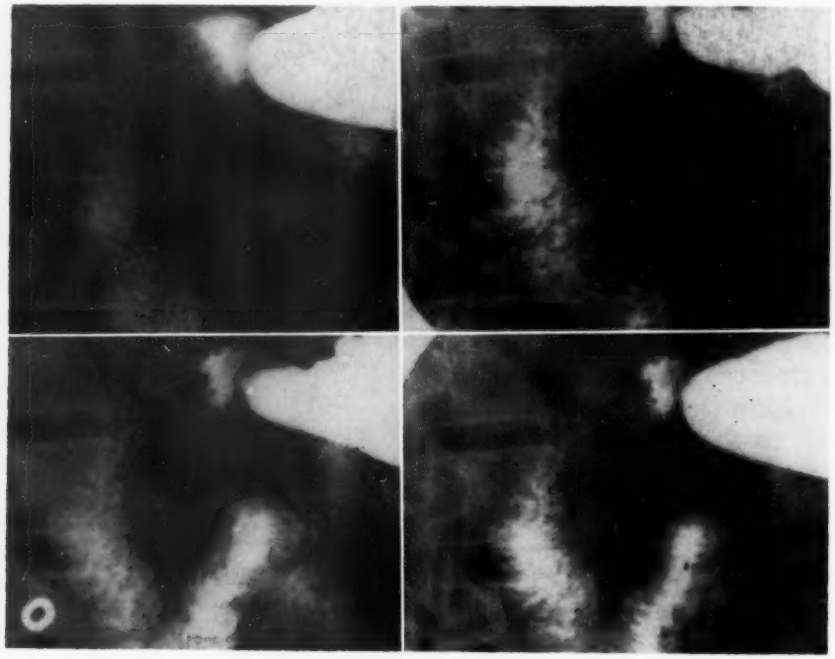


Fig. 19. R.A., a fifty-seven year old male, presented a history of jaundice and weakness of five weeks' duration. During the previous two weeks, he experienced an 8 pound weight loss. At another hospital a diagnosis of infectious hepatitis was made. Surgery revealed a carcinoma of the head of the pancreas. Roentgenograms show the flattening of the folds of the mesial lower second duodenum with a shallow indentation produced by the mass and marked spreading and ironing of the post-bulbar area caused by the enlarged common duct.

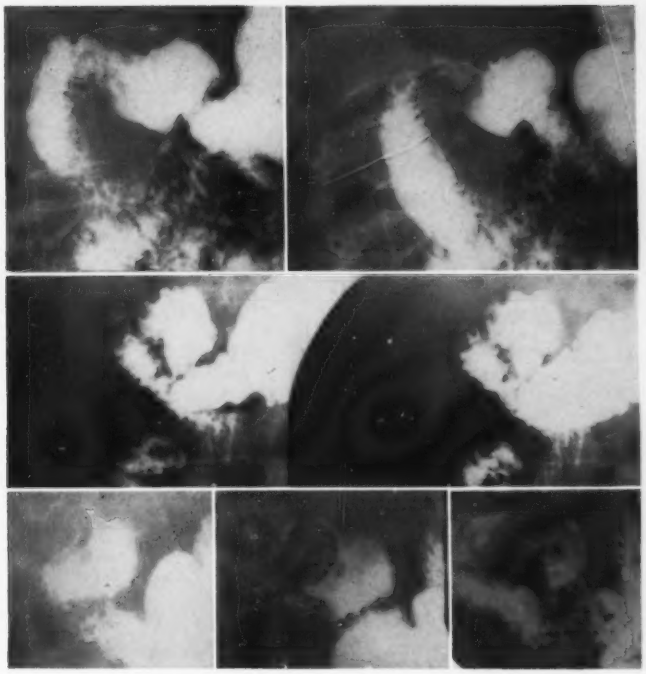


Fig. 20. M.E., a sixty-six year old male, presented a history of first experiencing intermittent epigastric discomfort three months previously. This became continuous, accompanied by a sensation of fullness. He also developed superficial phlebitis of both lower extremities. No jaundice was noted and no masses were felt. Surgery revealed carcinoma of the head of the pancreas. Roentgenograms show the double contour on the inner aspect of the duodenum with some irregularity of the folds at the lower inner aspect of its second part. In the prone position the mesial direction of the second part of the duodenum is demonstrated.

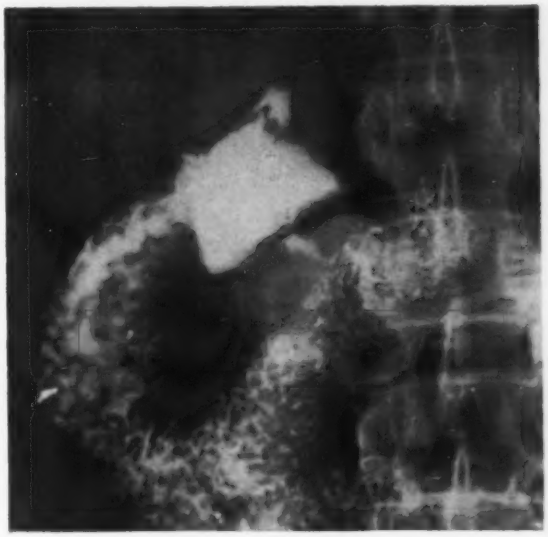


Fig. 21. S.L., a forty-three year old male, presented a history of ulcerative colitis at the age of seventeen. At the age of twenty-two, an ileostomy was performed and at the age of thirty-seven it was closed. In mid-June, 1958, he developed anorexia, progressive jaundice and by August had lost 19 pounds of weight. Surgery revealed tumor in the region of the papilla and the head of the pancreas. A radical pancreatico-duodenectomy was performed. Microscopic examination showed colloid carcinoma of the end of the pancreatic duct in the region of the papilla of Vater. Roentgenogram demonstrates the large irregular filling defect in the region of the papilla with some changes in the folds. (Courtesy of the Department of Radiology, Johns Hopkins Hospital and Medical School.)

Fig. 22. T.A., a forty-seven year old male, presented a history of jaundice and fatigue of ten days' duration. Surgery revealed a polypoid adenocarcinoma of the ampulla of Vater with involvement of the common bile duct.

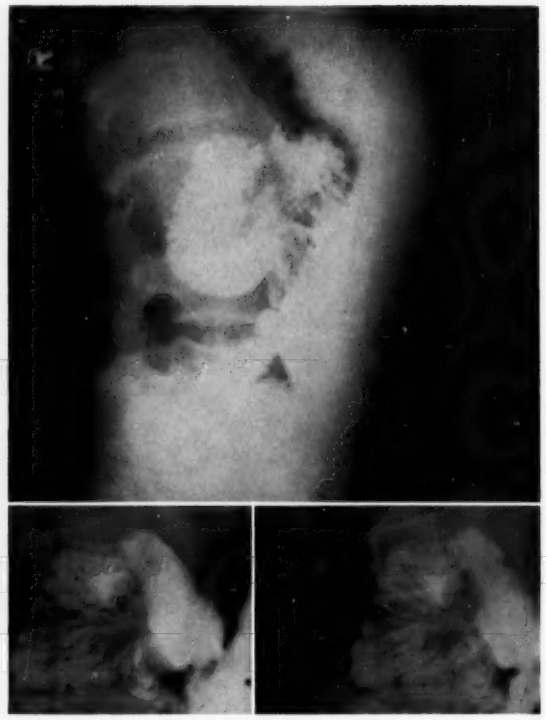


Fig. 23. This sixty-one year old male presented a history of marked weight loss during the previous five months and jaundice of two weeks' duration with bloating and fullness. Surgery revealed a carcinoma of the head of the pancreas with liver metastases. Roentgenograms show the inverted 3 (Frostberg) sign.

walls of the involved segment of the duodenum may be noticeable (Fig. 36). Later, this area may become narrowed due to fibrosis (Fig. 37). Patients with these lesions have a marked tendency to bleed as apparently the ulcer is often located near the pancreatico-duodenal artery which it erodes. Regional enteritis (Berk,[3] McGarity[19]) and tuberculosis (Fig. 38) of the duodenum have been described, but are rare. Usually both borders of the duodenum are involved and some segments irregularly stenosed, with skip areas and string signs.

C. Benign tumors of the duodenum might be due to a leiomyoma (Fig. 39), adenoma, fibroma, lipoma, hemangioma or

Fig. 25. Y.S., a fifty-six year old male, presented a history of sharp mid-thoracic pain radiating across the abdominal wall of five months' duration, increasing constipation of two months' duration, anorexia and fullness. There was no jaundice. Surgery revealed a carcinoma of the entire pancreas, invading the adjacent fundus of the stomach. Roentgenograms show the irregularity with fixation of the folds on the inner aspect of the second duodenum and the presence of a mass on the medial aspect of the fundus.

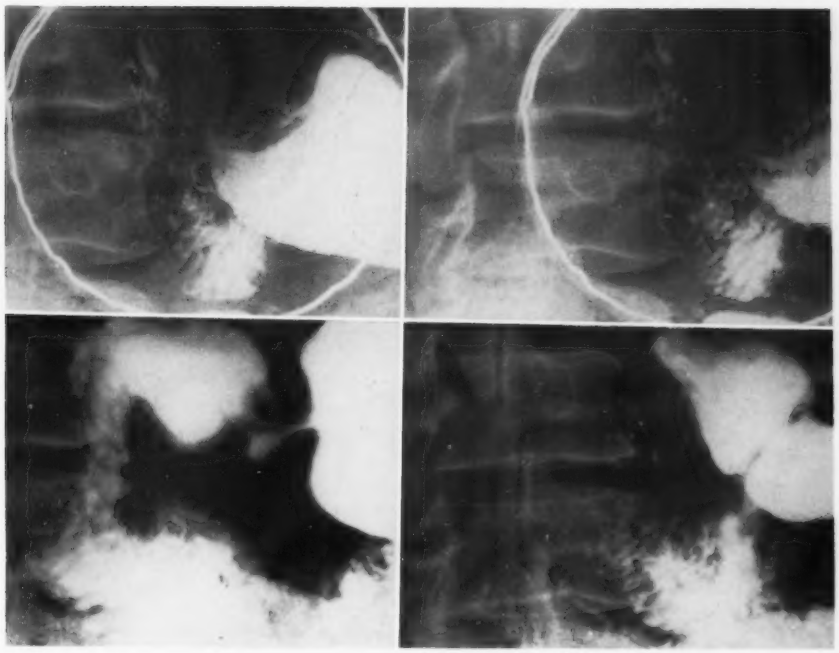
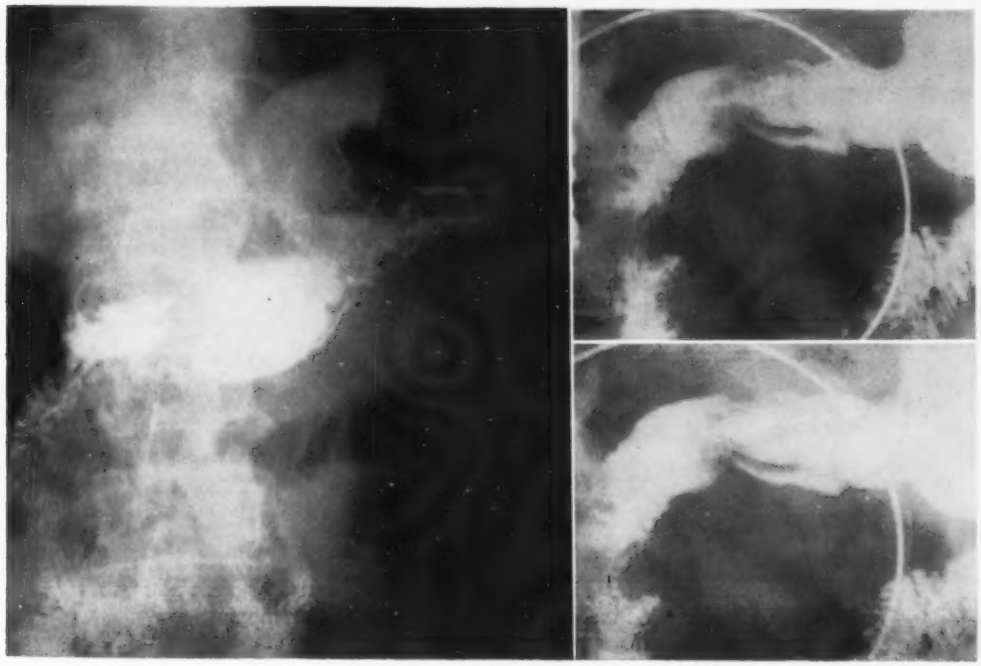


Fig. 24. H.E., a sixty-four year old male, presented a history of diarrhea, a 12 pound weight loss, jaundice of ten days' duration and dull, aching pain in the epigastrium. He had been a heavy drinker until the previous six years and was known to be a diabetic. A firm mass was felt in the left upper quadrant. Surgery revealed a carcinoma of the head and body of the pancreas. Roentgenograms show the irregularity, fixation of folds on the inner aspect of the duodenum and marked irritability of the folds with a skeletonized appearance. The pressure roentgenograms show the antrum to be compressed by the neoplasm. This was also noted in lateral views.



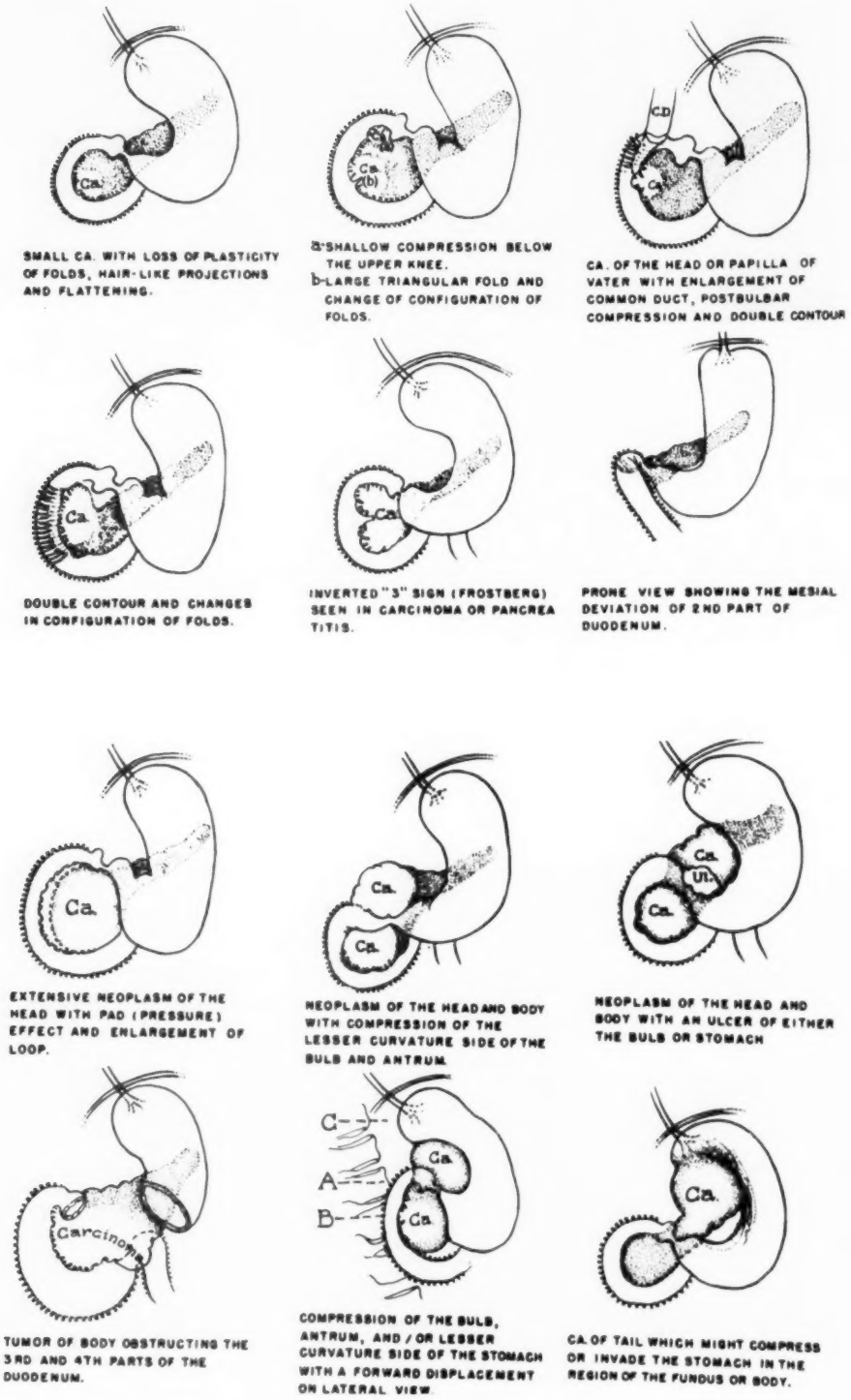

Ca.
SMALL CA. WITH LOSS OF PLASTICITY OF FOLDS, HAIR-LIKE PROJECTIONS AND FLATTENING.
Ca.
(b)
a-SHALLOW COMPRESSION BELOW THE UPPER KNEE.
b-LARGE TRIANGULAR FOLD AND CHANGE OF CONFIGURATION OF FOLDS.
C.D
Ca.
CA. OF THE HEAD OR PAPILLA OF VATER WITH ENLARGEMENT OF COMMON DUCT, POSTBULBAR COMPRESSION AND DOUBLE CONTOUR
Ca.
DOUBLE CONTOUR AND CHANGES IN CONFIGURATION OF FOLDS.
Ca.
INVERTED "3" SIGN (FROSTBERG) SEEN IN CARCINOMA OR PANCREATITIS.
PRONE VIEW SHOWING THE MESIAL DEVIATION OF 2ND PART OF DUODENUM.
Ca.
EXTENSIVE NEOPLASM OF THE HEAD WITH PAD (PRESSURE) EFFECT AND ENLARGEMENT OF LOOP.
Ca.
Ca.
NEOPLASM OF THE HEAD AND BODY WITH COMPRESSION OF THE LESSER CURVATURE SIDE OF THE BULB AND ANTRUM.
Ca.
Ul.
Ca.
NEOPLASM OF THE HEAD AND BODY WITH AN ULCER OF EITHER THE BULB OR STOMACH
Carcinoma
TUMOR OF BODY OBSTRUCTING THE 3RD AND 4TH PARTS OF THE DUODENUM.
C
A
B
Ca.
Ca.
COMPRESSION OF THE BULB, ANTRUM, AND / OR LESSER CURVATURE SIDE OF THE STOMACH WITH A FORWARD DISPLACEMENT ON LATERAL VIEW.
Ca.
CA. OF TAIL WHICH MIGHT COMPRESS OR INVADE THE STOMACH IN THE REGION OF THE FUNDUS OR BODY.

FIG. 26. Drawings showing the different stages in development and roentgenologic manifestations of pancreatic carcinoma.

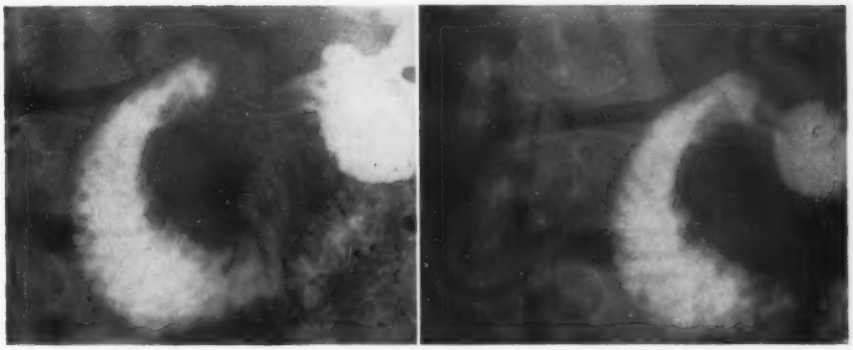


Fig. 27. Ch.M., a twenty-three year old female, presented a history of abdominal pain and vomiting of one and one-half days' duration. At the age of seventeen she began to have sharp mid-epigastric pain radiating to the back. The clinical diagnosis was pancreatitis.

aberrant pancreas. These are usually smooth and produce a discrete filling defect without involvement of the mucosa.

D. A stone lodged within the ampulla of Vater may produce a filling defect with concomitant jaundice (Fig. 40). Ascaris lumbricoides if nestled in the duodenum may show as a filling defect, but none of these roentgen findings could be mistaken for lesions caused by a pancreatico-ampullary neoplasm. Intramural hematomas have a distinctive pattern (Felson and Levin[9]). In the presence of adhesions of the second and third parts, irregularities of the involved duodenal wall may be noticeable (Ochsner[22]).

III. *Extrahepatic biliary tract and gallbladder neoplasms*, if of sufficient size, usually compress the lesser curvature side of the antrum and upper knee and/or descending lateral segment of the duodenum (Fig. 41). Rarely, a large sized tumor of the gallbladder produces a lateral displacement of the descending duodenum that is indistinguishable from a pancreatic carcinoma (McConnell[18]). Choledochal cysts may produce a large pressure defect on the duodenal loop with displacement and also compression of the antrum (Liebner[17]).

IV. *Primary tumors of the liver* usually present no diagnostic problem except that occasionally pancreatic carcinomas may be responsible for large metastases in the liver, or, conversely, liver tumors may metastasize to peripancreatic lymph nodes, and the picture becomes complicated.

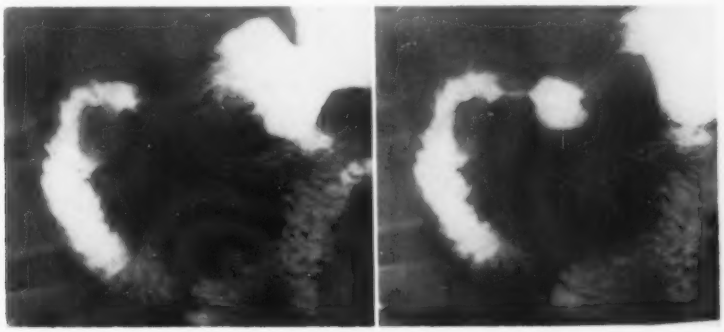


Fig. 28. G.Dm., a thirty-nine year old male, presented a history of intermittent abdominal pain of five years' duration which had recently become severe. The pain started in the left upper quadrant and radiated to the shoulder. The clinical diagnosis was pancreatitis. The roentgenologic pattern of tuberculosis in these cases is sometimes indistinguishable from that of pancreatic carcinoma.

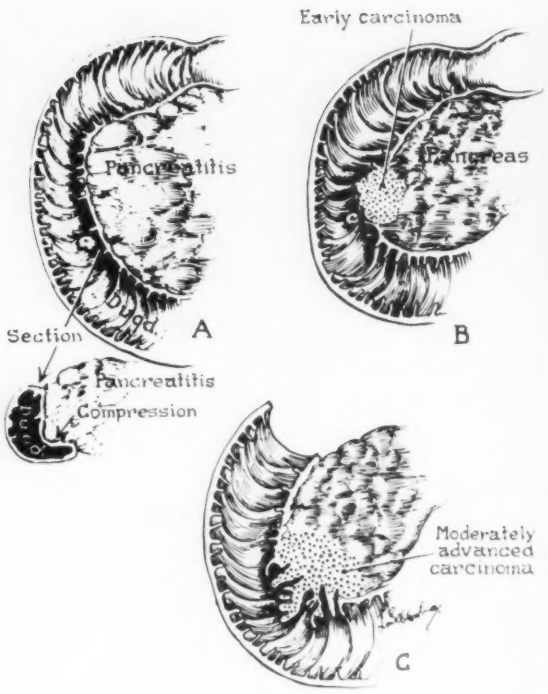


Fig. 29. Drawings show the differential diagnostic patterns of pancreatitis (*A*) and pancreatic carcinoma (*B* and *C*).

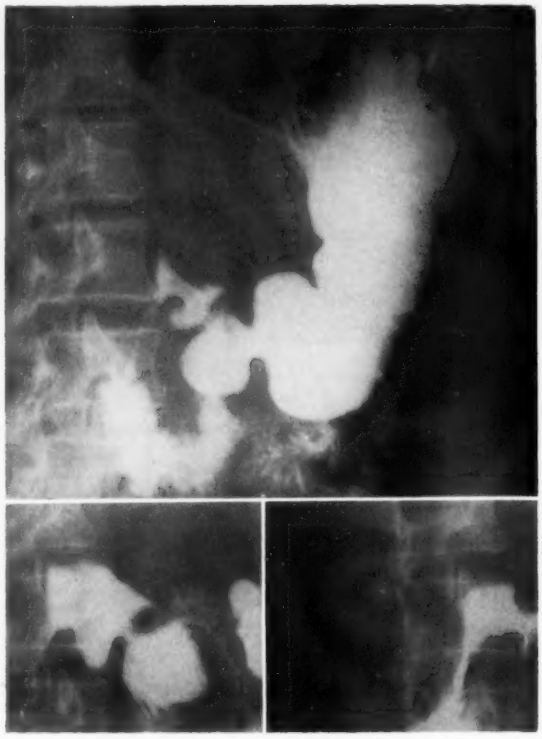


Fig. 30. S.H., a thirty-one year old female, presented a history of sharp right upper quadrant pain of nine days' duration. Surgery revealed chronic cholecystitis and cholelithiasis with chronic pancreatitis.

V. *Retroperitoneal masses* or nodular involvement may occur in primary new growth (sarcoma, lymphoblastoma), metastases or inflammatory processes like tuberculosis or infectious mononucleosis. These retroperitoneal masses at the pancreatic level might produce large pressure defects on the inner aspect of the duodenal loop, or, if nodular involvement is present, multiple scalloped indentations (Poppel[24]). Usually, different surfaces of the duodenum are involved, and the uninvolved area may be completely normal. These masses can be detected on lateral views. In the right prone

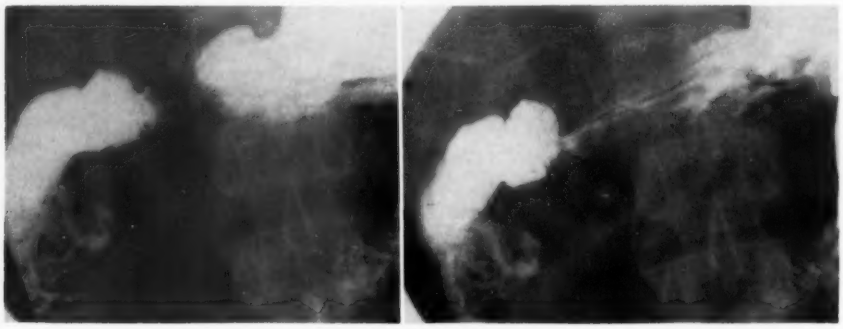


Fig. 31. K.L., a nineteen year old male, presented a history of sharp abdominal pain of eighteen hours' duration and occasional "spasm" of the stomach. Surgery revealed a pancreatic cyst with necrotizing pancreatitis. Roentgenograms show a fist-sized mass in the head of the pancreas.

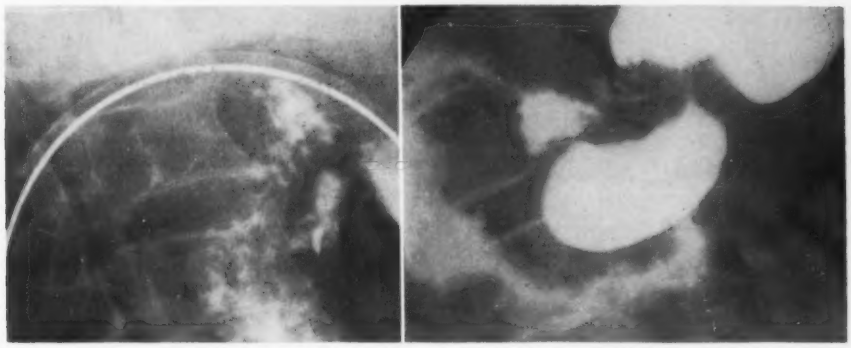


Fig. 32. G.B. presented a history of headache and deep sleep with stertorous respirations. On admission the blood sugar was 57 mg. per cent; after twenty-four hours of fasting, it was 40 mg. per cent. Surgery revealed an islet cell adenoma of the head of the pancreas. Roentgenograms show the compression of the inner aspect of the duodenum below the papillary segment.

anterior oblique position they may imitate tumors of the head of the pancreas, and the differential diagnosis becomes difficult (Fig. 42).

VI. *A large aneurysm of the anterior wall of the abdominal aorta or a hemorrhage around the head of the pancreas* due to a perforation of a vessel may produce a pressure defect on the inner aspect of the sweep (Fig. 43). Also, *lipomatosis* around the head of the pancreas may produce an enlargement of the duodenal loop.

CONCLUSION

1. With recent advances in pancreaticoduodenal surgery an improvement in the long term cure rate in cases of pancreaticoampullary carcinoma is possible, and, therefore, an early diagnosis is of great importance.

2. In the initial stages of the disease the clinical history might not be too distinctive, but some of the symptoms are highly significant and should arouse the suspicion of the radiologist.

3. Early changes on the inner aspect of the duodenal loop can often be detected if a careful examination of this area is conducted, and, thus, a high rate of preoperative diagnostic accuracy can be obtained by roentgenologic procedures.

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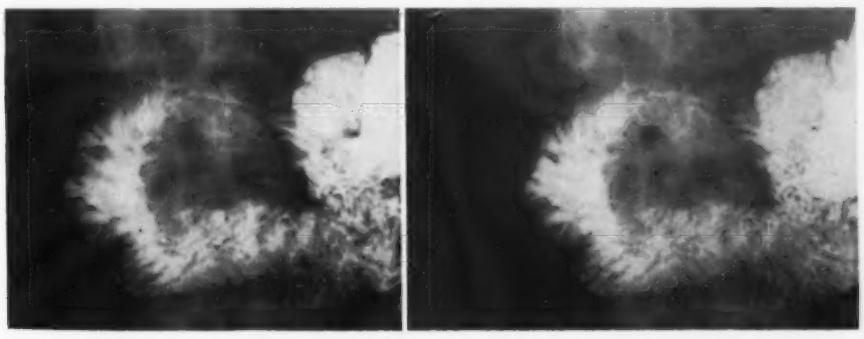

Fig. 33. D.H., a sixty-eight year old male, presented a history of a melanocarcinoma of the scalp with metastases to the cervical lymph nodes three years previously and nausea and a 7 pound weight loss of a few months' duration. Autopsy revealed metastatic melanocarcinoma involving skin, lungs, pancreas, spleen, liver, stomach, adrenal glands and lymph nodes. Roentgenograms show the irregular compression of the inner aspect of the duodenal loop.

FIG. 34. W.J., a sixty-nine year old white male, presented a history of sharp right upper quadrant pain of four months' duration with constipation for several months. Surgery revealed a fungating carcinoma of the lower second and third parts of the duodenum. Roentgenograms show complete destruction of the mucosa, filling defects, rigidity and a narrowed lumen.

FIG. 35. R.D., a forty-three year old male, presented a history of dull abdominal pain, especially in the right lower quadrant, of eight weeks' duration, with nausea and vomiting and a 32 pound weight loss. Surgery revealed 6 large tumor masses, one in the ileocecal area and 5 along the course of the small intestine with enlarged lymph nodes along the duodenum and intestine. The diagnosis was lymphoblastoma. Roentgenogram shows the irregular narrowing of the entire duodenum; the fourth segment seems to contain a polypoid mass. (Courtesy of the Department of Radiology of the Johns Hopkins Hospital and Medical School.)

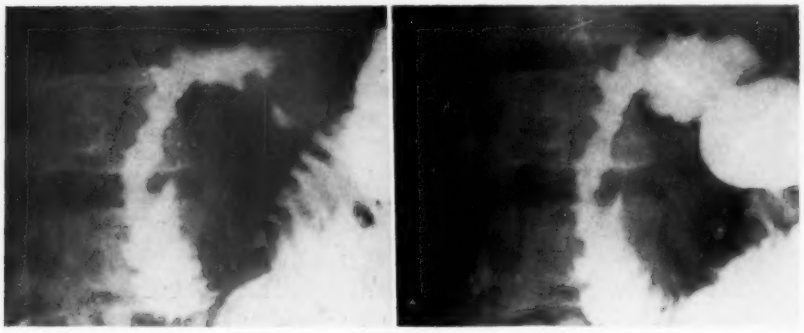

FIG. 36. D.B., a forty-two year old male, presented a history of persistent epigastric pain and occasional melena. Gastrointestinal series prior to admission were reported as negative. Surgery revealed an ulcer of the second part of the duodenum. Roentgenograms show the marked irritability and slight narrowing of the upper half of the second part of the duodenum.

FIG. 37. Roentgenogram shows the constricted postbulbar area and the double contour caused by the enlarged head of the pancreas. In this case surgery revealed ulcer and pancreatitis.

FIG. 38. J.A., a seventeen year old female, presented a history of nine hospital admissions for tuberculosis. The clinical diagnosis was tuberculous enteritis and peritonitis with fistulous tracts between the stomach and small intestine. (Courtesy of the Department of Radiology of the Johns Hopkins Hospital and Medical School.)

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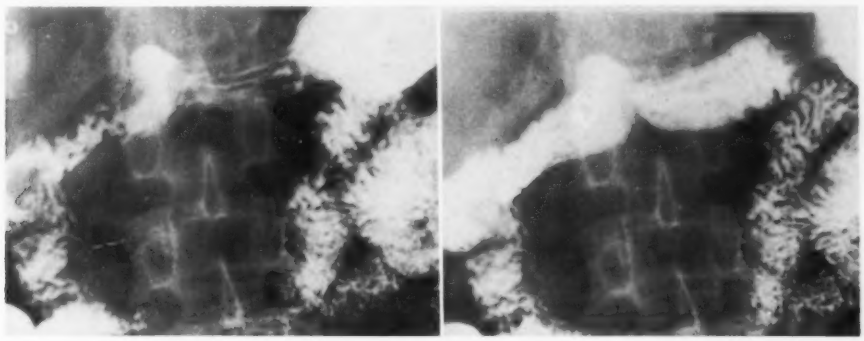

FIG. 39. P.D., a sixty year old male, presented a history of vague abdominal pain of four years' duration and a slightly tender epigastrium. Surgery revealed a tumor, 3 cm. in diameter, in the ampullary area of the duodenum. The pathologic report was leiomyoma.

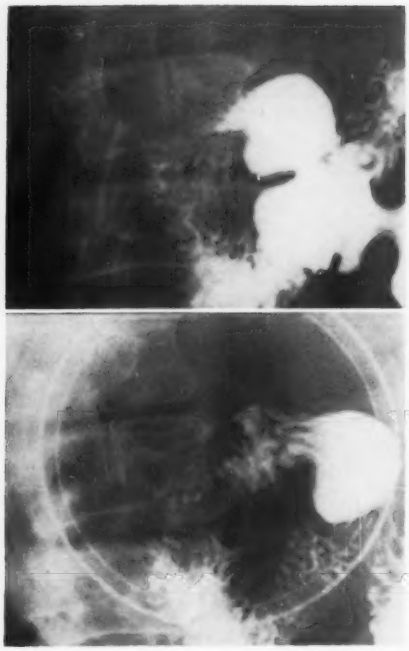

FIG. 40. G.H., a seventy-one year old male, presented a history of abdominal pain of three months' duration and recurrent jaundice. Surgery revealed an oval stone in the papilla with the common duct markedly dilated. Roentgenograms show a defect-like pattern in the ampullary area.

FIG. 41. S.E., a fifty-three year old female, presented a history of the sudden onset of jaundice and mild mid-thoracic and epigastric pain of two weeks' duration. Surgery revealed a carcinoma of the gallbladder. Roentgenograms show the compression of the lateral aspect of the duodenum.

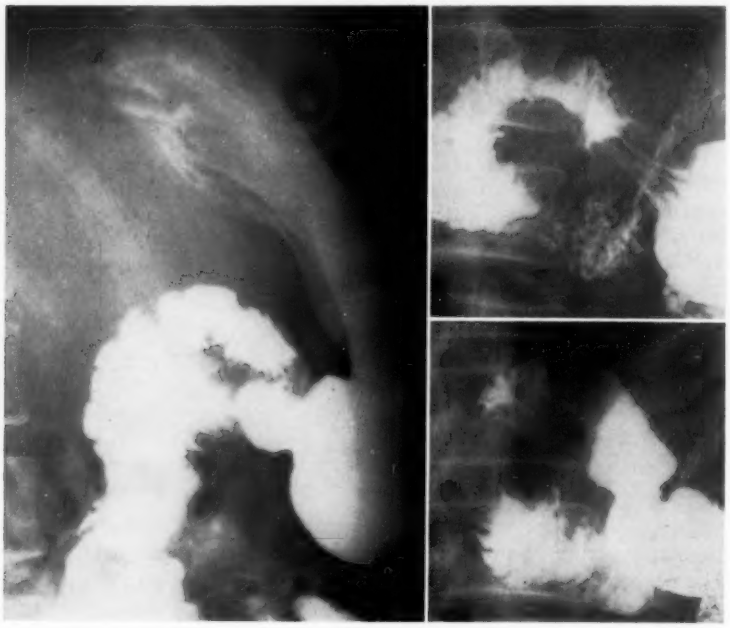

Fig. 43. M.E., a seventy-six year old male, was admitted as an emergency patient with sharp laminating pain in the left upper quadrant. He died twelve days later. At autopsy a mass of clotted blood surrounded the head of the pancreas and the second part of the duodenum, a result of perforation of the superior mesenteric artery. Roentgenogram shows the compression of the duodenum. During fluoroscopy the obstruction of the second duodenum was quite impressive.

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Fig. 42. F.T., a forty-five year old male, presented a history of loss of weight, anorexia and abdominal pain. A hard mass was palpable in the left upper quadrant. Surgery revealed an undifferentiated malignant lymphoma of the retroperitoneal tissues with involvement of the mesenteric and pancreatic lymph nodes.

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Vol. 86, No. 1

VISCERAL "AIR LUMENOGRAM"

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A CAREFUL study of the radiolucent visceral air shadows on the plain roentgenogram or other roentgenograms of the abdomen will occasionally reveal organic lesions of the gastrointestinal tract. This paper reports 9 cases in which signs suggestive of gastric peptic ulcer disease, gangrenous appendicitis, and neoplasm of the stomach and colon were evident on the plain roentgenograms of the abdomen. The significant changes were seen retrospectively except in 3 cases—a gastric lymphoma (Case v), a gangrenous appendicitis (Case iv), and a gastric ulcer (Case i). However, with more careful examination of the roentgenograms, the diagnosis might have been suggested by the preliminary and ancillary abdominal studies (*e.g.*, the cholecystograms, excretory urograms, etc.). We have not attempted to determine from our material the percentage of cases in which such findings will be visualized on the plain roentgenogram of the abdomen; it is obvious that it would be extremely difficult to obtain such figures.

The visceral gas pattern or outline is routinely studied in those cases presenting clinical evidence of intestinal obstruction, perforation of a viscus, or an intra-abdominal mass. Roentgenograms obtained incident to the study of the urinary collecting system, the lumbosacral spine, etc. usually are not adequately scrutinized for the intestinal gas pattern.

As early as 1917, Carman and Miller[1] noted variations in the size or the configuration of the stomach fundus gas bubble. They pointed out that the normally regular and symmetric outline of the gas bubble might be broken by a splenic or hepatic tumor. They also noted that an intrinsic tumor, involving the cardia and, usually, the lesser curvature side, produced an opaque deformity in this aspect of the gastric bubble. Kirklin[4] later stressed the importance of this manifestation and, in an analysis of 68 cases of tumor of the cardia of the stomach, he noted that a mass projecting into, or producing a defect in, the gas bubble was seen on routine chest roentgenograms in about 50 per cent of the cases.

The stomach gas bubble normally presents a hemispheric radiolucent outline below the left hemidiaphragm. A polypoid new growth projecting into the lumen of the stomach will produce a deformity of this gas bubble.

We are presenting 9 cases in which there was a consistent and persistent distortion of the configuration of a gas filled viscus on two or more roentgenograms, and have designated the term "air lumenogram" as a descriptive label for such visceral changes observed on the plain abdominal roentgenograms. These cases are submitted to stimulate a wider interest in the closer evaluation of the abdominal gas pattern.

GASTRIC PEPTIC ULCER

In 3 cases of peptic ulcer disease, the ulcer was clearly outlined by air on a plain roentgenogram of the abdomen. The lesions measured from 1.4 to 3.0 cm. in their greatest diameter, and were proven by surgical exploration.

In Case i (Fig. 1, *A* and *B*), the ulcer was visualized on a plain roentgenogram of the abdomen obtained eight days prior to performing an upper gastrointestinal series. In Case ii (Fig. 2, *A* and *B*), the "air lumenogram" of the ulcer was visualized on a roentgenogram made one month following the upper gastrointestinal studies.

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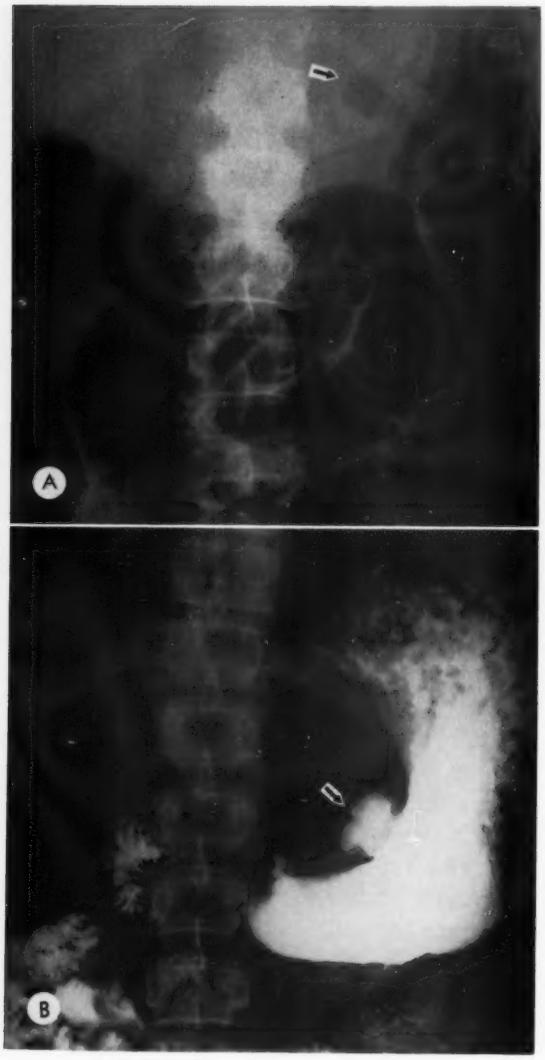


FIG. 1. Case I. (*A*) The large gastric ulcer niche (arrow) on the lesser curvature is sharply delineated, outlined by air. Even the meniscus of mucosal thickening can be discerned. The patient, a forty-two year old male alcoholic, admitted February 21, 1957, was referred from the emergency room in an intoxicated condition and with a history of gastrointestinal bleeding for several hours. This roentgenogram was obtained subsequent to the upper gastrointestinal series. (Courtesy of Dr. Marvin Silverstein, Atlanta, Georgia.) (*B*) Upper gastrointestinal roentgenogram reveals a large, penetrating, peptic gastric ulcer (arrow) and a duodenal ulcer (the latter better demonstrated on other roentgenograms). Surgery substantiated the roentgen findings.

In Case III (Fig. 3, *A*, *B* and *C*), the ulcer was visualized on a barium enema roentgenogram made seven days subsequent to an upper gastrointestinal series.

The configuration of the air shadow of the ulcer and its position, as observed in these cases, are believed to be sufficiently characteristic for at least a presumptive diagnosis.

GANGRENOUS APPENDICITIS

Fischer[2] recently presented 1 case of a dilated, fixed, gas filled appendix with an air fluid level indicative of a gangrenous, nonperforated appendix. It has been well documented that the normal appendix may contain air only intermittently.[3] A non-

FIG. 3. Case III. (*A*) Upper gastrointestinal roentgenogram, February 18, 1959. The recurrent ulcer of the lesser curvature is in precisely the same location as the gastric ulcer seen in 1956. On surgical exploration, a 3×2 cm. benign peptic ulcer was found on the lesser curvature. (*B*) Barium enema study, February 25, 1959. Note the round, sharply defined radiolucent area in the left upper abdominal quadrant, lying medial to the splenic flexure and cephalad to the air-filled gastric antrum (arrow). (*C*) Line drawing of *B* showing air outlining the gastric ulcer.

This forty-four year old male had initial symptoms of abdominal pain in 1942. In 1956, roentgen examination revealed a gastric ulcer of the lesser curvature, 10 mm. in diameter, which, with subsequent medical management, showed healing roentgenographically. Six months prior to the above upper gastrointestinal studies, he experienced recurrence of severe abdominal pain, frequent vomiting, and a thirty pound weight loss.

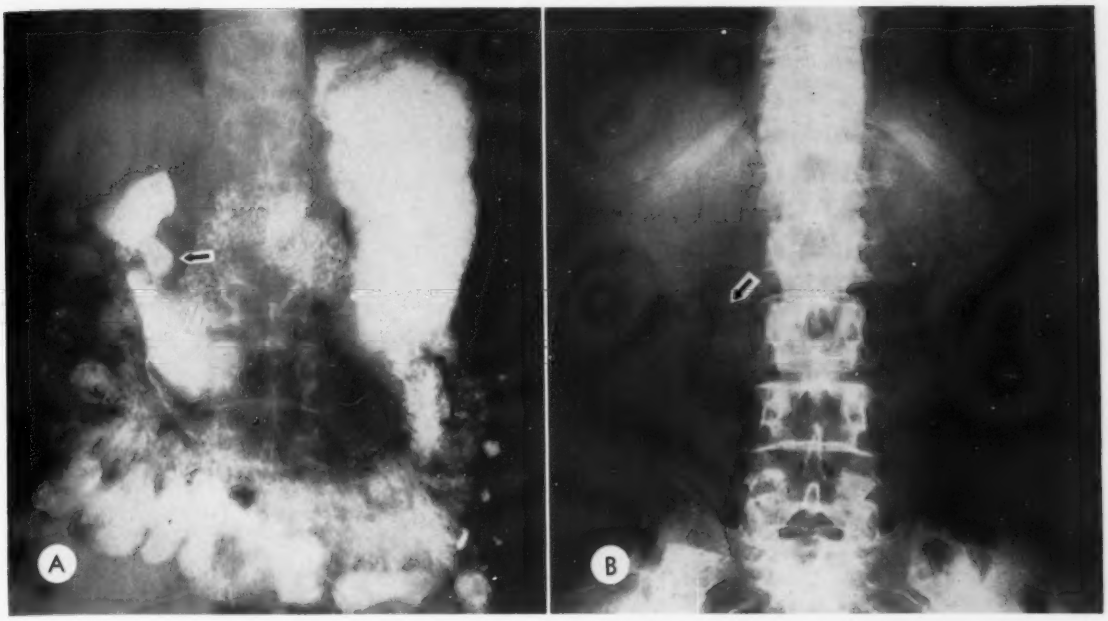
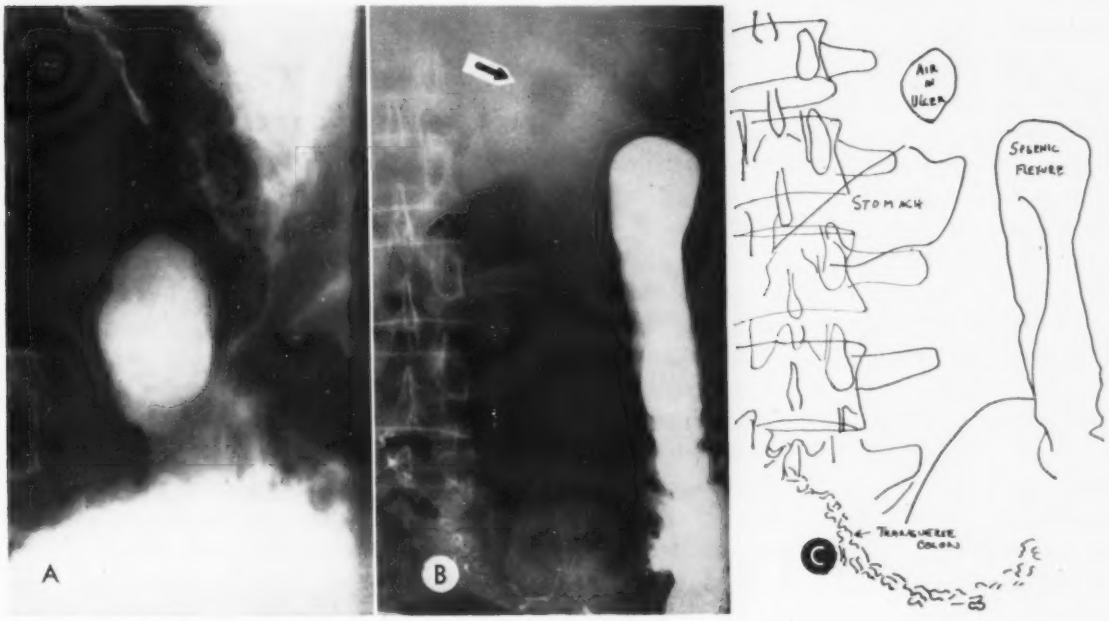


Fig. 2. Case II. (*A*) Upper gastrointestinal roentgenogram, May 25, 1959, demonstrates a large ulcer crater (arrow) of the prepyloric area. This was also observed on two previous gastrointestinal examinations with the oral administration of barium. (*B*) Roentgenogram, taken on readmission, June 26, 1959, when surgery was proposed for partial obstruction and intractable pain secondary to pyloric ulcer, shows an ulcer crater of the pylorus outlined by air (arrow). Surgical exploration revealed a 1.4 cm. by 0.2 cm. deep prepyloric ulcer along the lesser curvature of the stomach. There was no evidence of neoplasm on pathologic examination. (Courtesy of Dr. Joseph Chang, Emory University Hospital, Atlanta, Georgia.)



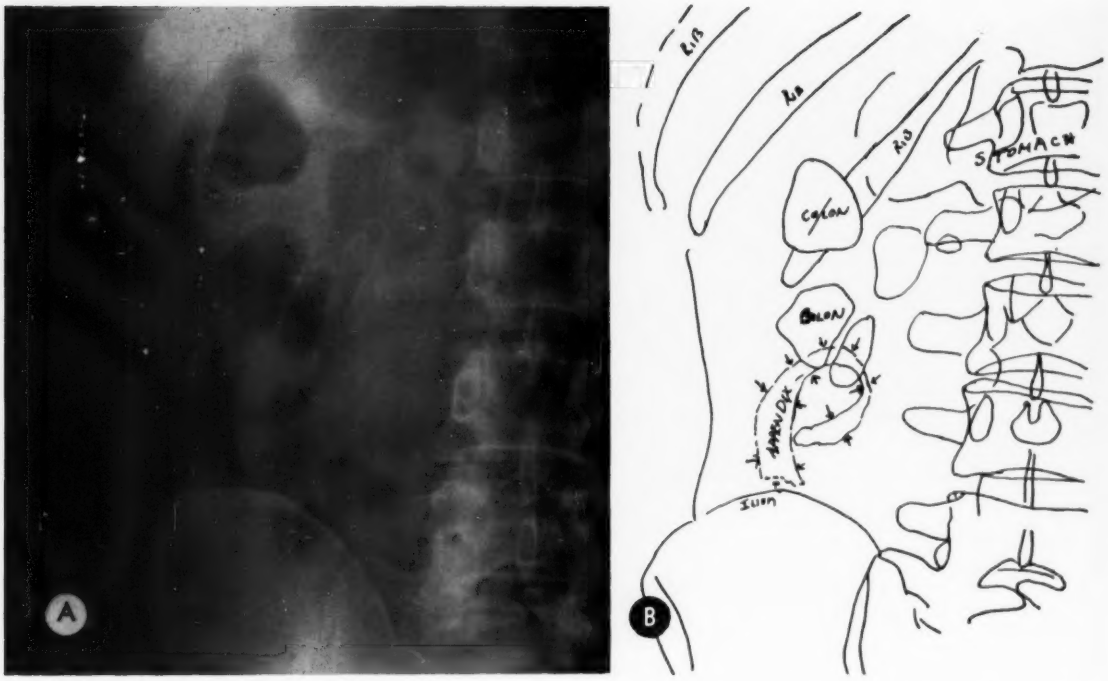


FIG. 4. Case IV. (*A*) Flat roentgenogram of the abdomen, January 3, 1959. Air outlining the lumen of the appendix is well visualized. (*B*) Line drawing of *A*.

The patient, a thirty-two year old white male and a diabetic for thirteen years, had onset of abdominal pain three days prior to the date of this study with marked tenderness over the right lower quadrant on light palpation. At appendectomy, performed on January 3, 1959, a purulent gangrenous perforated appendix was found with a large, adjacent, inflammatory mass lying retrocecally along the lateral peritoneal wall. Culture of the appendix revealed aerobacteria and numerous gram positive spores; the types were not determined on subsequent cultures.

pathologic appendix in the subhepatic area or in the presence of distention of the right colon, as in paralytic ileus, may contain gas without a fluid level.

Case IV, a thirty-two year old white diabetic male, had a gangrenous, perforated appendicitis. The appendiceal lumen on the flat roentgenogram of the abdomen was sharply outlined by air (Fig. 4, *A* and *B*). Unfortunately, an upright roentgenogram of the abdomen was not obtained so that the presence or absence of fluid and the degree of immobility of the appendix was not roentgenographically determined. Surgical exploration on the same day as the roentgen examination revealed a purulent, gangrenous, perforated appendix, adherent to an adjacent inflammatory mass. This would indicate that the air filled appendix can be seen in gangrenous appendicitis, even though perforation may have taken place.

NEOPLASM OF THE STOMACH

A case of Hodgkin's disease involving the cardia, fundus, and lower esophagus with invasion of the left hemidiaphragm showed a deformity of the cardia with a mass projecting into the stomach air bubble (Case V, Fig. 5, *A* and *B*). This was clearly visualized on the posteroanterior chest roentgenogram. The deformity was substantiated by an upper gastrointestinal series and by surgical exploration.

In another instance (Case VI, Fig. 6, *A*, *B* and *C*), an excretory urogram revealed a persistent abnormal deformity of the stomach outlined by air; this lesion was

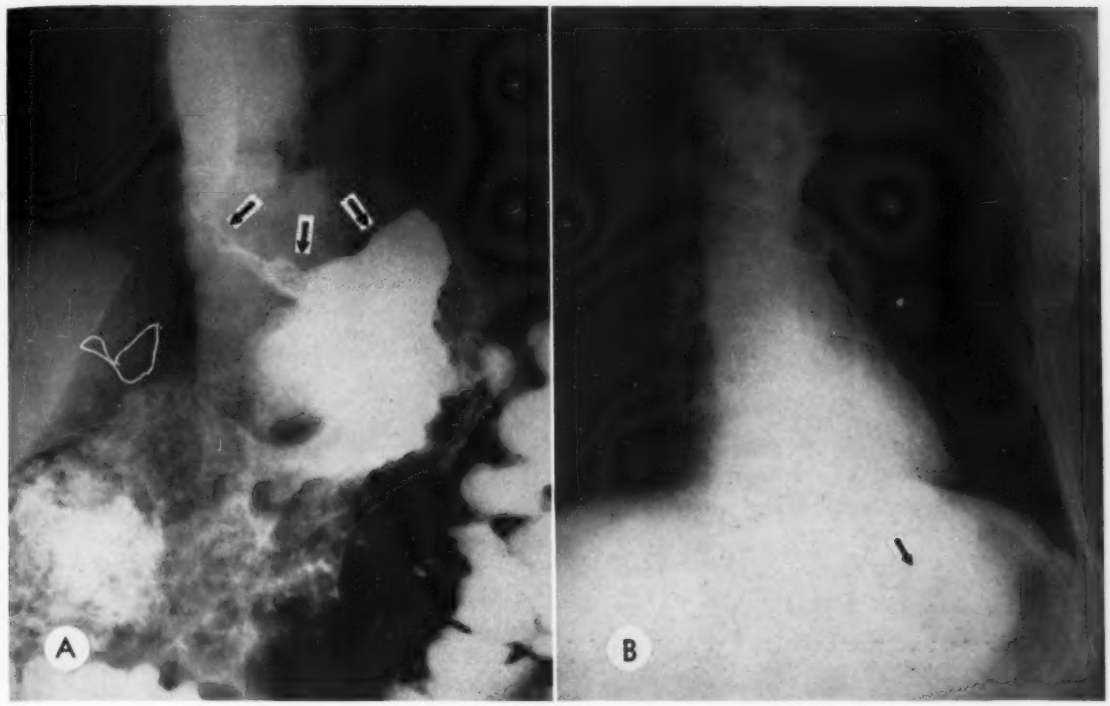


Fig. 5. Case v. (*A*) Upper gastrointestinal roentgenogram, February 12, 1959, of a fifty year old white male who had a history of weight loss, shows an extensive organic lesion (arrows) involving the cardia and fundus of the stomach and extending above the diaphragm to involve the distal esophagus. Surgical exploration revealed Hodgkin's disease of the stomach and distal esophagus with invasion of the diaphragm. (*B*) Posteroanterior chest roentgenogram, February 6, 1959, shows the neoplasm deforming the cardia and fundus of the stomach, with air outlining the inferior margin of the tumor mass.

confirmed by barium study. On abdominal exploration, an inoperable adenocarcinoma of the stomach was found.

NEOPLASTIC LESIONS OF THE COLON

Three cases are presented in which the abdominal roentgenograms demonstrated malignant lesions of the colon. In Case vii (Fig. 7, *A*, *B* and *C*), an excretory urogram revealed the typical finding of a lesion involving the ascending colon. A similar pattern was demonstrated on barium enema examination performed two days later. An operable carcinoma of the ascending colon was found at surgery.

The second case (Case viii, Fig. 8, *A*, *B* and *C*), a sixty-nine year old patient, was treated for genitourinary sypmtoms in December, 1957. On plain roentgenograms of the abdomen an irregular tapering of the proximal splenic flexure was observed at the site of a carcinoma. A roentgen diagnosis of this defect was not made until some time later, August 21, 1958, by means of a barium enema examination. A more careful study of the original plain roentgenograms would have led to an earlier diagnosis.

In the third patient (Case ix, Fig. 9, *A*, *B* and *C*), a plain roentgenogram of the abdomen obtained on April 26, 1959, revealed an intraluminal polypoid mass in the descending colon. This was definitively visualized, of course, on barium enema studies the following day.

SUMMARY

A gastric ulcer niche was demonstrated

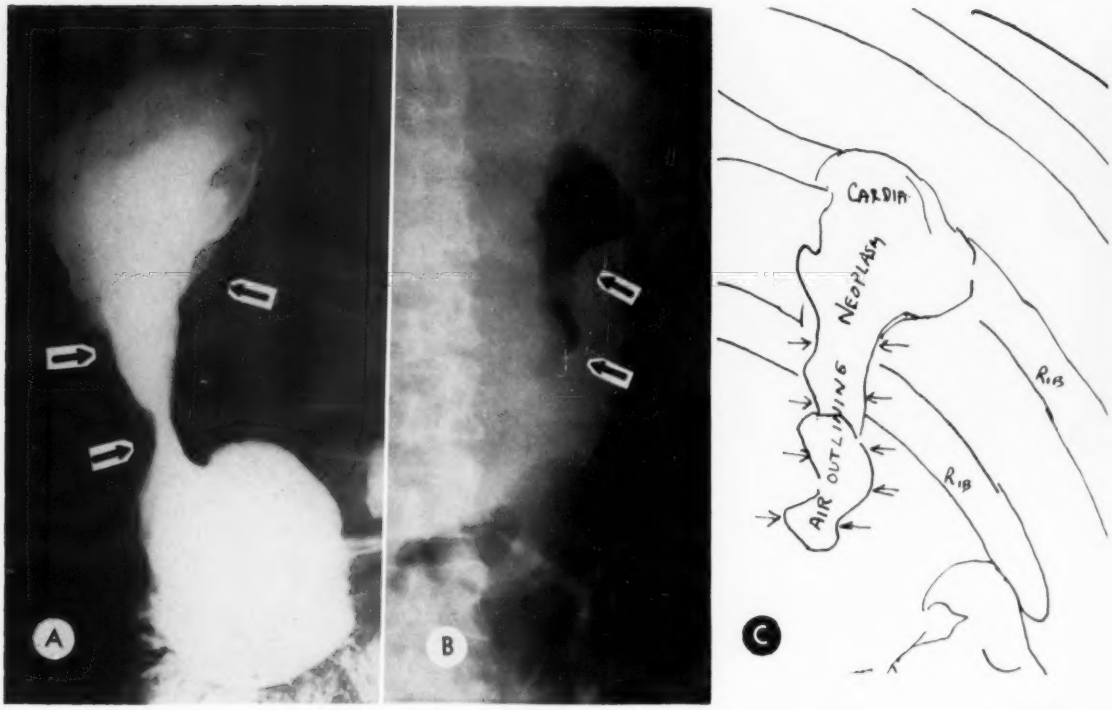


FIG. 6. Case VI. (*A*) Upper gastrointestinal roentgenogram, July 29, 1958. An annular, stenotic, rigid, organic process (arrows), apparently a neoplasm, is involving the greater and lesser curvature of the stomach. At abdominal exploration, an inoperable adenocarcinoma of the stomach metastatic to the omentum was found. (*B*) Flat roentgenogram of the abdomen, August 6, 1958, obtained during exploratory urography. Essentially the same stereotyped narrowed body of the stomach is seen outlined by air. (*C*) Line drawing of *B*.

This fifty-nine year old white male was well until four weeks prior to admission, July 25, 1958, when he developed an epigastric, gnawing pain and a slight, retrosternal, burning sensation.

in 3 cases on the plain roentgenograms of the abdomen; these ulcers were clearly defined.

A patient with gangrenous appendicitis revealed an appendiceal lumen sharply outlined by air. It is of interest that the fixed appendix was found to be perforated at the time of surgical exploration.

Two neoplasms of the stomach were visualized on roentgenograms of the chest and abdomen, respectively.

Three neoplasms involving the colon, (the ascending colon, splenic flexure, and descending colon) were clearly outlined by air on plain roentgenograms of the abdomen.

In only 3 of the above 9 cases (Hodgkin's disease of the stomach, an air-filled gangrenous appendix, and a gastric ulcer), was the diagnosis made from the plain roentgenogram. Nevertheless, the air outline or visceral "lumenogram" in each instance presented an appearance characteristic enough to have suggested the diagnosis.

Definitive gastrointestinal studies with contrast media are essential to confirm the impressions gained from the "air lumenograms." The extreme variability of the mucosal pattern or configuration of the stomach does not allow a presumptive diagnosis based only on a single plain roentgenogram of the abdomen. However, if a typically pathologic gastric pattern, not only of the fundus but also of the body, is visualized on the plain roentgenogram, it may be suggestive of the diagnosis.

Retrospective examination of the roentgenograms of several patients with neo-

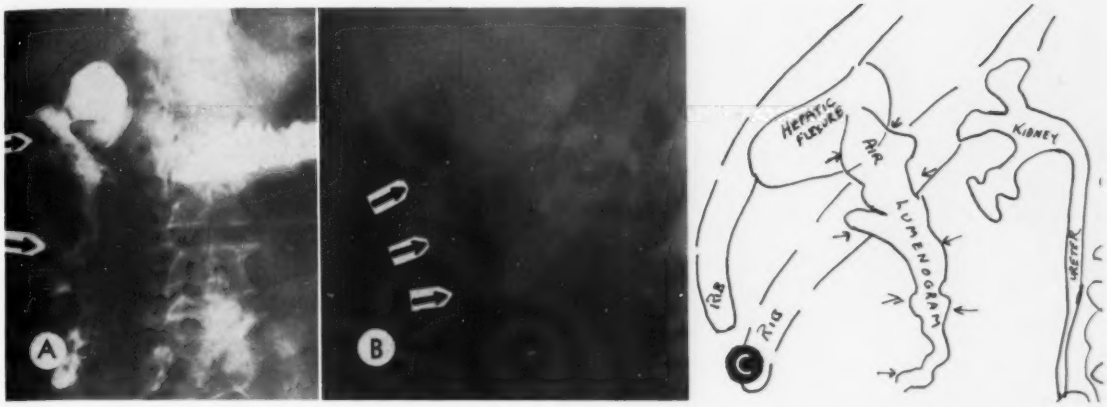


Fig. 7. Case VII. (*A*) Barium enema study, July 11, 1958, indicates an organic lesion (arrows) of the ascending colon measuring 8.5 cm. in length. This seventy year old Negro male four months prior to admission had noted right abdominal discomfort ("gas pains"). He presented with evident, moderate, recent, weight loss, anemia, and a palpable mass in the right upper quadrant. (*B*) Excretory urogram, July 9, 1958, shows displacement of the renal shadow toward the midline. The rigid, typical, long curvilinear contours outlined by air (arrows) represent the neoplasm of the ascending colon. (*C*) Line drawing of *B*. The arrows indicate the narrowed stenotic lumen of the ascending colon involved by neoplasm. At operation, a 9 cm. carcinoma of the ascending colon was found immediately adjacent to the hepatic flexure, with invasion of the posterior abdominal wall, the psoas muscles, and the vena cava.

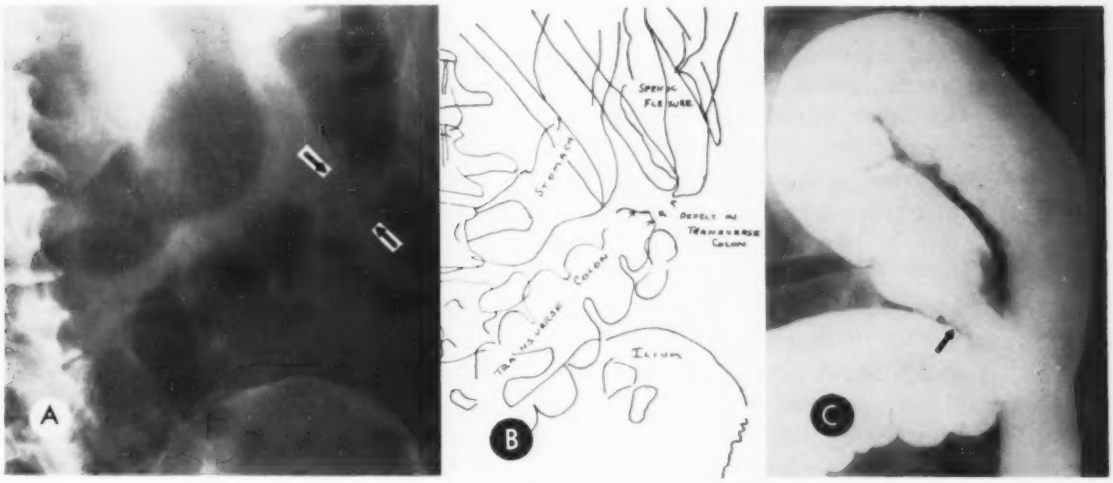


Fig. 8. Case VIII. This sixty-nine year old Negro male was on the Genitourinary Service from December, 1957, to March, 1958, for benign prostatic hypertrophy treated by transurethral resection. (*A*) Plain roentgenogram of the abdomen obtained during excretory urography, December 17, 1957. Arrows indicate an irregular tapering of the proximal splenic flexure caused by a carcinoma which was not diagnosed until later. Note also the absence of air at the site of the carcinoma, with moderate distention of the colon proximal to the lesion. (*B*) Line drawing of *A*. (*C*) Barium enema study, August 21, 1958, after the patient was readmitted for anorexia, marked weight loss, lower mid-abdominal pain and constipation. An adenocarcinoma of the transverse colon just proximal to the splenic flexure, measuring 4×5 cm., was found on surgery.

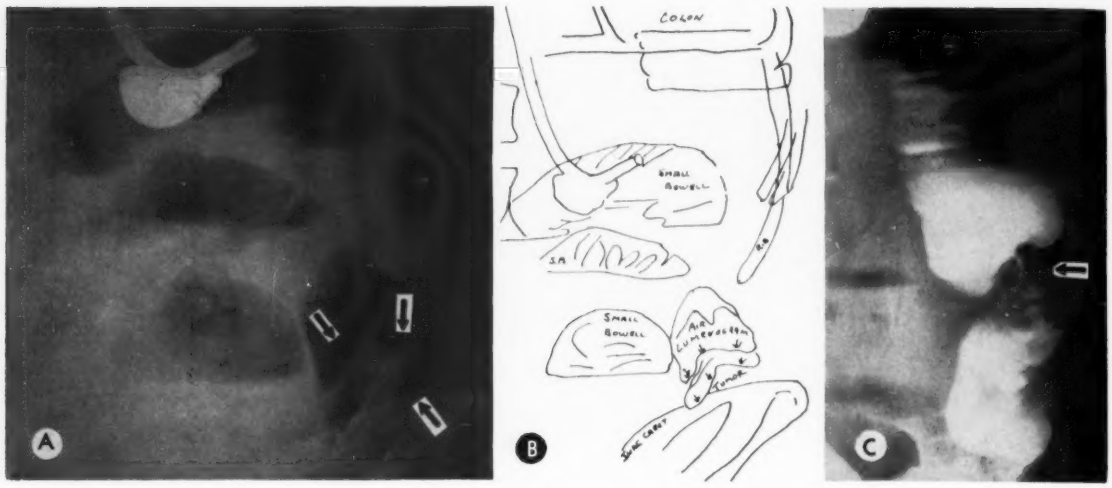


FIG. 9. Case IX. (*A*) Plain roentgenogram of the abdomen, April 26, 1959. The irregular polypoid or exophytic margins of the carcinoma of the descending colon project into the proximal air-filled segment of colon (arrows). Marked large and small bowel distention, with wide air-fluid levels in both, is seen. (*B*) Line drawing of *A*. The arrows indicate what is apparently the tumor mass of the descending colon projecting over the level of the left iliac crest. (*C*) Barium enema study, April 27, 1959. The stenotic lumen, caused by the annular carcinoma of the descending colon, is well demonstrated.

plasm of the stomach indicated an additional difficulty in visualizing the gastric "air lumenogram" due to the overlying lumbar spine which obscures the gas pattern on the frontal view.

The characteristic deformity in the air outline of the large bowel which is produced by a neoplasm may be of valuable ancillary assistance in the later evaluation of the barium enema studies.

CONCLUSION

Cases of various lesions of the gastrointestinal tract are presented in which the diagnosis was apparent on examination of the visceral gas patterns seen on plain abdominal roentgenograms, excretory urograms, roentgen studies of the lumbosacral spine, etc.) It is felt that a closer scrutiny of these roentgenograms will often lead to a presumptive diagnosis, which must then be confirmed by contrast studies. We do not believe that the "air lumenogram" can supersede the confirmatory gastrointestinal or barium enema studies, but it may, in some instances, serve as a valuable diagnostic aid.

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THE EXAMINATION OF PATIENTS WITH SUSPECTED PERFORATED ULCER USING A WATER-SOLUBLE CONTRAST MEDIUM*

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UNTIL the present time, when examining patients suspected of having a perforated peptic ulcer, the function of the radiologist has been confined to the demonstration of pneumoperitoneum. Nothing beyond this has been necessary because the usual treatment of perforated peptic ulcer has been the immediate surgical closure of the perforation. There is, however, a growing concept that for selected patients nonoperative treatment may be a more suitable form of therapy.[1,2,7,8,10] Wangensteen[11] is credited with first suggesting, in 1935, that certain patients with perforated peptic ulcer might be treated only by aspiration of the stomach. In 1939, Mullen[4] cautiously applied this method to a small series of patients. Since then, larger series have been reported both in England and in this country. In 1957 Taylor,[9] one of the earliest and strongest advocates of this form of therapy, reported 256 patients with perforated ulcer of whom 21 were treated surgically and 235 by aspiration. The mortality of those treated by aspiration was about 2.5 per cent. Seeley and Campbell[6] reported 139 cases treated conservatively from 1945 to 1953. There were 7 deaths, giving an overall mortality of 5 per cent, or, by excluding 5 inoperable patients, a corrected mortality of 1.5 per cent. The same authors in reviewing the literature found 857 nonoperative cases, in which the mortality was 11.4 per cent with a corrected mortality, excluding 73 inoperable cases, of 4.5 per cent. The mortality in 1,622 operative cases reviewed by them was 8.7 per cent. Heslop *et al.*[3] reported a mortality rate of 7.7 per cent in a series of 104 nonoperative cases as compared with 8.6 per cent in 104 operative cases. In the 8 nonoperative deaths, 4 occurred in patients unsuited for surgery.

The nonoperative management of perforated peptic ulcer is predicated on the knowledge that many perforations have sealed when the patient is first seen. Sealing of the perforation by the direct apposition of adjacent structures is frequently observed even at early laparotomy. However, it is most difficult to differentiate clinically between those perforations which have recently sealed and those which continue to leak. In addition, errors in diagnosis constitute a serious problem since other conditions which simulate perforated peptic ulcer must be identified promptly so that proper treatment may be instituted.

It is evident that it is extremely advantageous to have a roentgenographic method that would demonstrate the presence or absence of leakage with a high degree of accuracy and which would also aid in the identification of other conditions simulating perforated peptic ulcer. The radiologist has been understandably reluctant to administer barium sulfate for fear that it might spill into the peritoneal cavity. Although water-soluble contrast media have been used to examine the gastrointestinal tract with increasing frequency, we have encountered only one reference to their use in conjunction with perforated ulcer. Rea[5] reported in 1952 that he had administered 10 cc. of diodrast to 5 patients and ob-

* Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.
The contrast medium used in this study was supplied by Winthrop Laboratories, New York, New York.
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tained supine and upright roentgenograms of the abdomen. Diodrast was seen in the peritoneal cavity in 1 case.

Early in 1958, at the suggestion of one of the authors (C.J.B.), a study was begun to evaluate the incidence of spontaneous sealing of perforated peptic ulcers. A water-soluble contrast substance, hypaque, was instilled through a nasogastric tube as part of this study to determine roentgenologically whether or not sealing of the ulcer had occurred. It is the purpose of this paper to describe the technique of the examination and the roentgenographic findings in 120 patients. The clinical results of this study will be reported separately.

TECHNIQUE OF EXAMINATION

For a number of years in our department, the roentgenologic examination of each patient suspected of having a perforated intra-abdominal viscus has included an upright posteroanterior roentgenogram of the chest and two roentgenograms of the abdomen, consisting of an anteroposterior supine view and lateral decubitus view with the right side up. We believe a chest roentgenogram should be part of the examination of all patients with acute abdominal symptoms, not only to identify free subdiaphragmatic gas but also pulmonary or cardiac lesions which might be responsible for the patient's symptoms or complicate the situation.

Our first examination of the stomach and duodenum using a water-soluble contrast medium was fairly simple. After the preliminary roentgenograms had been obtained, 60 cc. of 50 per cent intravenous hypaque was instilled into the stomach through a Levin tube. Two anteroposterior roentgenograms of the abdomen were then made, one supine and the other with the patient in the semi-erect position. It soon became apparent that with this technique, leakage of fairly large amounts of hypaque into the peritoneal cavity could be detected easily. In one or two instances, the site of perforation was seen, but, in general, the stomach and duodenum were not well visualized. It was then decided to extend the examination, fluoroscoping each patient and obtaining spot roentgenograms if possible. This proved to be easily accomplishable and the examination soon developed into a modified but thorough upper gastrointestinal examination.

As noted above, our first examinations were performed with the intravenous preparation of hypaque. While this was quite satisfactory as a contrast medium, it was rather expensive since it was necessary to use at least two, and sometimes three vials. We were able to obtain powdered hypaque in bulk form and found, after some experimentation, that 50 gm. dissolved in 75 cc. of sterile water made a solution of ideal concentration. This volume has proved sufficient to perform most examinations. Several samples of powdered hypaque were examined bacteriologically. None contained any pathogenic bacteria. Ten drops of a wetting agent (tween 80) have been added to aid in the coating of the mucosa. All of the water-soluble contrast media are very bitter and cannot be administered orally unless a flavoring agent is added. We have not attempted to do this for such a substance might prove irritating to the peritoneal cavity. We did not believe it was necessary inasmuch as the stomach is emptied by nasogastric suction and the tube left in place in each patient suspected of having an acute abdominal lesion before the patient is brought to the radiology department.

The examination is now performed as follows: After the preliminary roentgenograms for pneumoperitoneum have been obtained, the patient who previously has been given a narcotic is placed on a fluoroscopic table and the solution of hypaque is given through the Levin tube. The patient is fluoroscoped carefully. If there is an obvious massive leak of contrast medium into the peritoneal cavity, spot roentgenograms are made rapidly to record the site of leakage and to determine, if possible, the nature of the lesion which has produced the perforation. Supine and semi-erect 14×17

inch roentgenograms are then made. If there is no massive leakage, the stomach and duodenum are examined in much the same manner as for a conventional upper gastrointestinal study. Multiple spot and the usual over-the-table Bucky roentgenograms are made, followed by supine and semi-erect 14×17 inch roentgenograms. The examination may be extended readily to include the small intestine when, for example, it becomes evident that the patient's symptoms are due to intestinal obstruction, etc.

Almost all the patients we have examined have been able to cooperate fully, even though many have been aged and critically ill. In only a few instances have we had to modify the study because of the patient's condition.

RESULTS

Patients suspected of having a perforated peptic ulcer, who are examined roentgenologically, while acutely ill, fall into the following groups: (1) gastroduodenal ulcer with or without leakage of contrast medium from the ulcer site, with pneumoperitoneum; (2) gastroduodenal ulcer with or without leakage of contrast medium from the ulcer site without pneumoperitoneum; (3) other conditions with pneumoperitoneum simulating perforated peptic ulcer, with or without gastroduodenal ulcer; and (4) other conditions without pneumoperitoneum simulating perforated peptic ulcer, with or without gastroduodenal ulcer.

For purposes of simplification, the term peptic ulcer has been used to include not only duodenal and benign gastric ulcer, but also malignant gastric ulcer. It was determined (Table 1) that 98 (82 per cent) of the 120 patients examined had a peptic ulcer which was responsible for their symptoms and 82 of these patients had a definite perforation in that there was pneumoperitoneum and/or leakage of contrast material from an ulcer. Excluding the 16 cases with neither pneumoperitoneum nor leakage, 93 per cent of the patients with perforated peptic ulcer had pneumoperitoneum and 50 per cent of the ulcers were leaking when examined. Of particular importance are the 6 patients, 7 per cent of those with proved perforated ulcer, who had leakage of contrast material from their ulcer in the absence of pneumoperitoneum.

TABLE I

SUSPECTED PERFORATED PEPTIC ULCER
120 PATIENTS
Males 99 (83%) Females 21 (17%)

Final Diagnosis—Peptic Ulcer—98 (82%)			
Males 83 (85%) Females 15 (15%)			
Pneumoperitoneum—no leakage			41 (42%)
Pneumoperitoneum—leakage			35 (36%)
No pneumoperitoneum—no leakage			16 (16%)
No pneumoperitoneum—leakage			6 (6%)
Final Diagnosis—No Peptic Ulcer—22 (18%)			
	Males	Females	
Pneumoperitoneum			
Diagnosis undetermined	2	1	
Perforated diverticulum of colon	1	4	
Perforated carcinoma of colon	—	1	
	3	6	9 (41%)
No Pneumoperitoneum			
Pancreatitis	3	—	
Cholecystitis	2	—	
Undetermined	2	—	
Perforated diverticulum of colon	1	—	
Traumatic rupture of jejunum	1	—	
Carcinoma of duodenum	1	—	
Intestinal obstruction	1	—	
Alcoholic gastritis	1	—	
Perforated appendix	1	—	
	13	—	13 (59%)

Twenty-two (18 per cent) of the 120 patients had some other conditions which clinically simulated perforated peptic ulcer. Of these 9 (41 per cent) had pneumoperitoneum and 13 (59 per cent) did not. The various diagnoses are listed in Table 1. It is of interest that 6 of the 9 patients with pneumoperitoneum were females while all 13 patients without pneumoperitoneum were males. This is perhaps due to the fact

TABLE II

LEAKAGE OF CONTRAST MEDIUM IN 82 PATIENTS WITH PROVED PERFORATION

		No Leakage	Leakage
Duodenal Ulcer—65 (Males 56, Females 9)			
Pneumoperitoneum	61 (94%)	33	28
No Pneumoperitoneum	4 (6%)	—	4
Total	65 (100%)	33 (50%)	32 (50%)
Benign Gastric Ulcer—14 (Males 9, Females 5)			
Pneumoperitoneum	13	8	5
No Pneumoperitoneum	1	—	1
Malignant Gastric Ulcer—3 (Males 3,* Females 0)			
Pneumoperitoneum	2	—	2
No Pneumoperitoneum	1	—	1

* Leakage in all 3 cases confined to area of perforation.

that perforation of a colonic diverticulum, the second most common cause of pneumoperitoneum, in our experience is more frequent in females than males.

Tables II and III summarize the occurrence and extent of leakage of contrast material from the 3 categories of gastroduodenal ulcer and the relationship of pneumoperitoneum to leakage. Leakage was found to occur in 4 distinct patterns: (1) generalized, spreading throughout all or most of the peritoneal cavity; (2) confined to the right upper quadrant or the liver edge; (3) confined to the left upper quadrant; and (4) confined to the immediate area of perforation.

Duodenal Ulcer. There were 65 patients with proved perforated duodenal ulcer, 61 (94 per cent) of whom had pneumoperitoneum. Leakage of contrast material occurred in 32 of these patients or 50 per cent of those with subsequently proved perforation. In almost one-half of these 32 patients, the leage was generalized (Fig. 1, *A* and *B*). In 10 patients it was confined to the right upper quadrant or liver edge (Fig. 2, *A* and *B*); and, in 7, the leakage was localized to the area of perforation (Fig. 3, *A* and *B*). Leakage occurred in 4 cases in which there was no demonstrable pneumoperitoneum, and in one of these, the leakage was generalized (Fig. 4, *A*, *B* and *C*). There were no cases in which leakage from the perforated duodenal ulcer was confined to the left upper quadrant of the abdomen.

TABLE III

EXTENT OF LEAKAGE OF CONTRAST MATERIAL

	Duodenal Ulcer		Benign Gastric Ulcer		Malignant Gastric Ulcer	
	Pneumoperitoneum	No Pneumoperitoneum	Pneumoperitoneum	No Pneumoperitoneum	Pneumoperitoneum	No Pneumoperitoneum
Generalized	14	1	3	—	—	—
Right Upper Quadrant	8	2	—	—	—	—
Left Upper Quadrant	—	—	2	1	—	—
Area of Perforation	6	1	—	—	2	1
Total	28	4	5	1	2	1

Benign Gastric Ulcer. Of the 14 cases of benign gastric ulcer, all of which were perforated, 13 had pneumoperitoneum. Leakage occurred in 6 cases; it was generalized in 3 instances and in the remainder, one of which did not have pneumoperitoneum, it was confined to the left upper quadrant (Fig. 5, *A*, *B* and *C*). There were no cases in which the leakage was localized to the right upper quadrant or in the area of perforation.

Fig. 1. Twenty year old male with acute symptoms of 2 hours' duration and no previous symptoms. Pneumoperitoneum was noted. Hypaque studies showed (*A*) a duodenal ulcer with massive leakage and urinary tract excretion and (*B*) an ulcer crater on the spot roentgenogram (arrow). Final diagnosis: duodenal ulcer with 1.0 cm. open perforation (surgery).

Fig. 2. Fifty-seven year old male with acute symptoms of 12 hours' duration and no previous symptoms. Pneumoperitoneum was noted. Hypaque studies showed (*A*) a duodenal ulcer tract along the lower edge of the liver, leakage confined to the right upper quadrant and (*B*) two days later, persistence of leakage. Final diagnosis: duodenal ulcer with 8 mm. open perforation (surgery).

Age and Sex. No relationship could be demonstrated between the age or sex of the patient and leakage of contrast material. The age and sex of those in whom leakage was demonstrated was not signifi-

FIG. 3. Forty year old male with acute symptoms of 6 hours' duration and previous symptoms of 10 months' duration. Pneumoperitoneum was noted. Hypaque studies showed (*A*) a duodenal ulcer crater with leakage confined to the area of perforation (arrow) and (*B*) a collection of hypaque (arrow) adjacent to the ulcer crater on the spot roentgenogram. Final diagnosis: duodenal ulcer with localized perforation (elective surgery).

cantly different from that of the entire group with perforated ulcer.

Duration of Symptoms. It was thought that the duration of ulcer symptoms or symptoms of acute perforation might have some bearing on whether the perforation would have sealed spontaneously by the time the examination was performed, *i.e.*, an acute ulcer might be expected to seal spontaneously more readily than a chronic ulcer. As shown in Table IV, there were 10 patients with perforated duodenal ulcer who had no previous ulcer symptoms; in 6 of these leakage was noted. On the other hand, no leakage could be demonstrated in 21 of the 41 patients with perforated duodenal ulcer who had ulcer symptoms for more than six months.

The length of time between the onset of acute symptoms of perforation and the roentgen examination of the stomach and duodenum is shown in Table V. Slightly more than one-half the patients with perforated duodenal ulcer examined within six hours and a somewhat smaller proportion of those examined from six to twelve hours after the onset of symptoms had leakage when examined. However, there was no significant difference in the percentage of unsealed ulcers found in patients examined either twelve to twenty-four hours or even more than forty-eight hours after the onset of symptoms.

The much smaller group of benign gastric ulcers does not appear to differ significantly from the perforated duodenal ulcer group in the relationship of duration of ulcer symptoms and symptoms of acute perforation to spontaneous sealing.

Malignant Gastric Ulcer. In distinct contrast were the 3 cases of malignant gastric ulcer. Leakage was present in all 3 of these cases, one of which did not have pneumoperitoneum, and in each the leakage was confined to the area of perforation (Fig. 6, *A* and *B*).

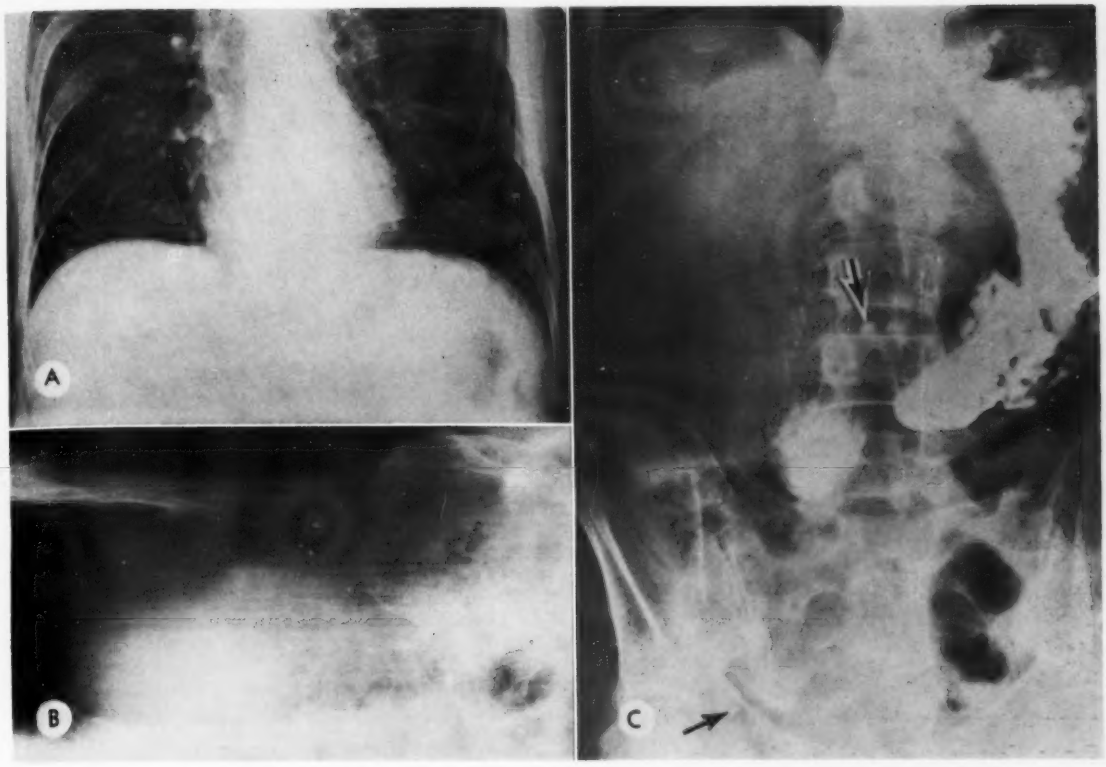

Fig. 4. Fifty-four year old male with acute symptoms of 2 hours' duration and previous symptoms of 7 days' duration. Upright chest and lateral decubitus roentgenogram showed (*A* and *B*) no evidence of pneumoperitoneum. Hypaque study revealed (*C*) that the contrast material coated the bowel in the right lower quadrant and pelvis (lower arrow). Upper arrow points to the ulcer crater. Final diagnosis: duodenal ulcer with 0.5 cm. open perforation (surgery).

Table IV

RELATION OF ULCER SYMPTOMS TO LEAKAGE OF CONTRAST MATERIAL

Duration of Symptoms (mo.)	No Leakage	Generalized Leakage	Leakage in Right Upper Quadrant	Leakage in Left Upper Quadrant	Leakage at Area of Perforation
		Duodenal Ulcer			
0	4	2	4	—	—
0–3	7	3	1	—	2
3–6	—	—	—	—	—
6	21	10	5	—	5
No History	1	—	—	—	—
		Benign Gastric Ulcer			
0	—	1	—	—	—
0–3	4	2	—	—	—
3–6	—	—	—	—	—
6	4	—	—	2	—
No History	—	—	—	1	—

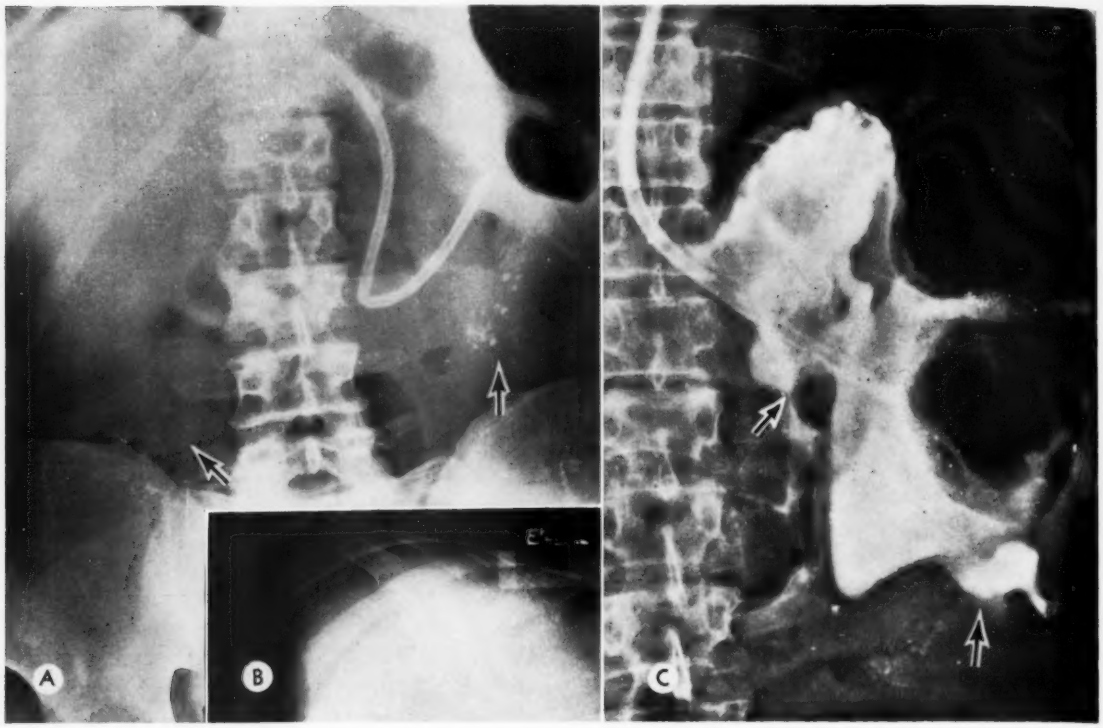

FIG. 5. Forty-seven year old male with no available history. (*A* and *B*) Loculated gas in the left upper quadrant was thought at first to be in the splenic flexure of the colon. Arrows point to pancreatic calculi. Differential diagnosis: pancreatitis versus perforated ulcer. (*C*) Hypaque study shows large gastric ulcer (upper arrow) with massive leakage (lower arrow) confined to left upper quadrant. Final diagnosis: perforated 2.5 cm. lesser curvature gastric ulcer.

TABLE V

RELATION OF DURATION OF SYMPTOMS OF PERFORATION TO LEAKAGE OF CONTRAST MATERIAL

Duration of Symptoms (hr.)	No Leakage	Generalized Leakage	Leakage in Right Upper Quadrant	Leakage at Area of Perforation
		Duodenal Ulcer		
0–6	14	9	4	4
6–12	11	1	2	2
12–24	6	3	1	1
24–48	0	1	1	0
48	2	1	2	0
		Benign Gastric Ulcer		
0–6	1	1	—	—
6–12	2	—	1	—
12–24	2	2	—	—
24–48	—	—	1	—
48	3	—	—	—
No History	—	—	1	—

Urinary Tract Excretion. Excretion of opaque material through the urinary tract was observed in several cases in which there was generalized leakage of contrast material into the peritoneal cavity. Hypaque is not appreciably absorbed from the gastrointestinal tract. The absorption presumably occurred through the peritoneum.

Diagnoses. All examinations have been included except several performed early in the development of the procedure which were technically unsatisfactory. Since modifying our technique to more closely resemble a conventional upper gastrointestinal examination and overcoming our reluctance to examine this type of patient, the studies have improved rapidly in quality and at present are rather uniformly good. All the examinations have been performed by the resident staff.

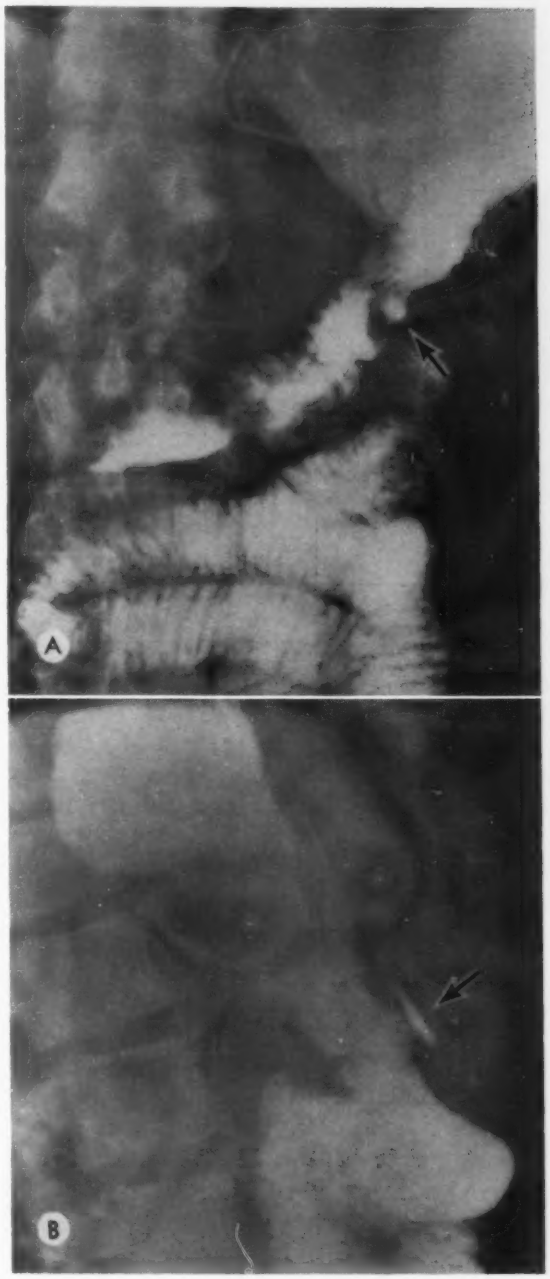

Fig. 6. Eighty year old male with acute symptoms of 1 day's duration and previous symptoms of 9 months' duration. Pneumoperitoneum was demonstrated. Hypaque studies showed (*A*) a greater curvature ulcer with surrounding filling defect (arrow) and (*B*) a small anterior localized perforation (arrow). Final diagnosis: malignant gastric ulcer with localized perforation.

Table VI indicates the manner of arriving at a final diagnosis. Each case is listed only once under the most reliable means of confirmation. All but 15 cases were confirmed by surgery (immediate or elective), follow-up gastrointestinal examinations, or autopsy. In these 15 patients, the final diagnoses were based on the hypaque studies and other roentgenologic examinations and quite unequivocal clinical findings.

There were 4 errors in roentgenologic diagnosis (Table VII). While the absence of leakage was successfully demonstrated in these cases, they were considered errors to the extent that the duodenal and gastric ulcers were not demonstrated by the hypaque studies. A sealed perforation was confirmed by surgery in 3 cases (Fig. 7, *A* and *B*) and in the fourth, a follow-up gastrointestinal examination demonstrated a duodenal ulcer.

COMMENT

The technique described in this paper of using a water-soluble contrast medium to examine the stomach and duodenum of a patient suspected of having a perforated peptic ulcer was developed in conjunction with the nonoperative management of perforated peptic ulcer and was prompted by the difficulty of recognizing clinically those cases in which the perforation continues to leak. The authors of previously published reports dealing with the conservative management of perforated peptic ulcer have assumed that all patients included had a defi-

Table VI

CONFIRMATION OF DIAGNOSIS

	Surgical Findings*	Follow-up Gastrointestinal Studies	Clinical Findings	Autopsy Findings
Ulcer	73	12	12	1
No Ulcer	12	5	3	2

* Includes cases confirmed by elective surgery.

TABLE VII

ERRORS IN DIAGNOSIS (4 CASES)

Sex	Age	Duration of Acute Symptoms (hr.)	Duration of Previous Symptoms	Pneumoperitoneum	Hypaque Study	Final Diagnosis
M	31	14	0	Yes	Normal, no leakage	Giant bleeding duodenal ulcer, sealed perforation (surgery)
M	50	10	0	No	Normal, no leakage	Duodenal ulcer (follow-up gastrointestinal study)
M	49	10	5 yr.	Yes	Normal, no leakage	Gastric ulcer, sealed perforation (follow-up gastrointestinal study and surgery)
M	30	2	2 wk.	Yes	Normal, no leakage	Gastric ulcer, sealed perforation (surgery)

nite perforation. However, there are those patients who develop acute symptoms simulating perforation strongly enough to be diagnosed as such without perforation actually being present. There were 16 patients (15 with duodenal ulcer and 1 with malignant gastric ulcer) in the present study who had neither pneumoperitoneum nor leakage of contrast material. It is quite likely that these cases represent penetrating rather than perforated ulcers.

The danger of misdiagnosis has been of considerable concern to all. In the present series, 18 per cent of the patients proved to have some condition not related to peptic ulcer. Some of these patients required prompt surgical intervention. It is obvious that the criteria used for selecting patients for the examination will determine the proportion of nonulcer patients in any given group such as ours.

Leakage was demonstrated in almost 50 per cent of the 65 cases of perforated duodenal ulcer, in a slightly smaller number, 43 per cent (6 of the 14 cases) of perforated benign gastric ulcer, and in all 3 cases of perforated malignant gastric ulcer. All patients were examined as soon after admission to the hospital as feasible and all those with generalized intraperitoneal leakage were operated upon promptly. However, for one reason or another, surgery was delayed in several patients with localized leakage. Two of these patients were reexamined. One had a perforated malignant gastric ulcer with leakage confined to the area of perforation. Two days later, the ulcer was shown to have sealed (Fig. 8, *A* and *B*). The other patient had a perforated duodenal ulcer with the leakage confined to the right upper quadrant and a well-defined tract along the liver edge. Reexamination three days later showed continued leakage from the ulcer and the patient was operated upon (Fig. 2, *A* and *B*). It should be stressed that of those examined more than twelve hours after the onset of symptoms, about one-half were found to have leakage.

It has been suggested by Taylor[9] and Seeley and Campbell,[6] among others, that acute ulcers, particularly in young individuals, can be expected to seal spontaneously and that perforated chronic ulcers often do not. Our results do not bear this out. The proportion of ulcers leaking at the time of examination was the same for patients with a history of ulcer symptoms of long duration as for those with a history of short duration. The incidence of leakage

did not vary significantly with the time which had elapsed between the onset of symptoms of perforation and the performance of the roentgen examination. This suggests that, perhaps, those perforations which are going to seal spontaneously do so very soon after the perforation has occurred.

FIG. 7. Thirty-one year old male with acute symptoms of 14 hours' duration and no previous symptoms. Pneumoperitoneum was noted. Hypaque studies showed (*A*) no leakage and (*B*) a normal duodenal bulb on spot roentgenogram. Final diagnosis: giant bleeding duodenal ulcer (delayed surgery).

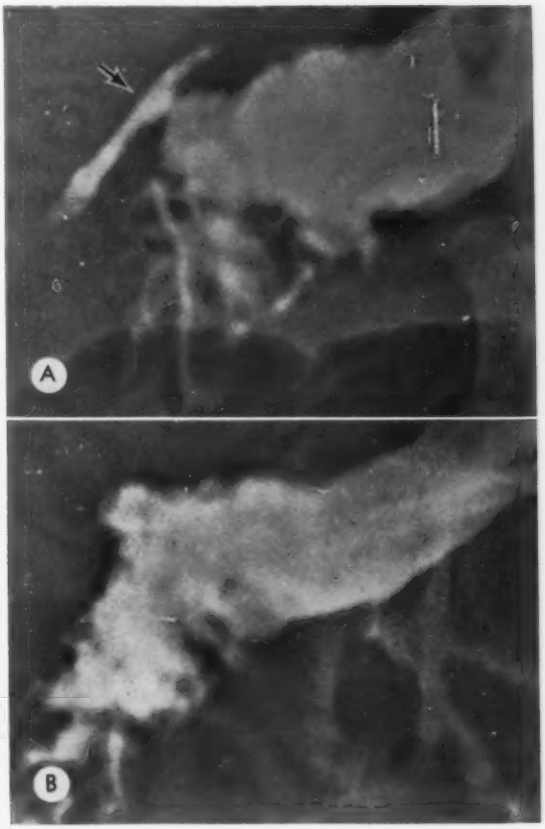

FIG. 8. Seventy-one year old male with acute symptoms of 36 hours' duration and previous symptoms of 1 year's duration. Pneumoperitoneum was noted. Hypaque studies showed (*A*) a large irregular gastric ulcer (arrow) with leakage confined to the area of perforation and (*B*) a sealed perforation on a repeat examination 2 days later. Final diagnosis: malignant gastric ulcer with sealed perforation (elective surgery).

Perforation of peptic ulcer in the absence of demonstrable pneumoperitoneum is particularly difficult to diagnose clinically, and even more difficult to prove. Possibly some of the 16 patients with neither pneumoperitoneum nor leakage had "forme fruste" perforations, though to be conservative we have assumed that they were not perforated. However, there were 6 patients without pneumoperitoneum with leakage of the ulcers at the time of examination; 4 from duodenal ulcers and 1 each from benign and malignant gastric ulcers. What is of considerable importance is that

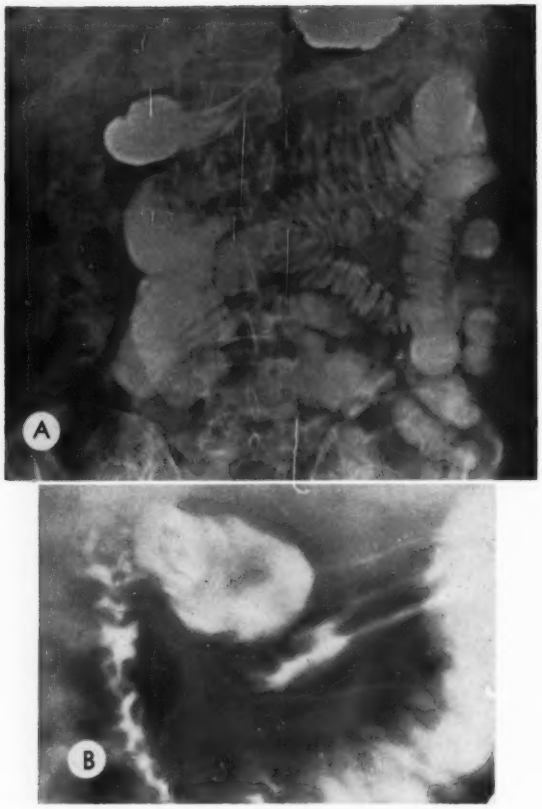

Fig. 9. Forty-eight year old male with acute symptoms of 24 hours' duration and no previous symptoms. Pneumoperitoneum was noted. Hypaque studies showed (*A*) a normal stomach and duodenum and no leakage and (*B*) a normal duodenal bulb on spot roentgenogram. Final diagnosis: perforated diverticulum of colon (surgery).

in one of these patients with an unsealed perforated duodenal ulcer the leakage was generalized.

The distribution and pathways of leakage of gastrointestinal contents from perforated peptic ulcer have long been known from surgical and postmortem observations. The procedure described in this paper has afforded the first opportunity to demonstrate these pathways visually. At the time of examination, generalized leakage throughout the peritoneal cavity was present in almost one-half of the patients with leaking duodenal and gastric ulcers. Leakage from perforated duodenal ulcers is frequently confined to either the right upper quadrant, the liver edge, or the area of perforation and from perforated benign gastric ulcer to the left upper quadrant. No localization to the right upper quadrant or area of perforation was observed in the small series of perforated benign gastric ulcers. It is also of interest that the leakage in all 3 cases of perforated malignant gastric ulcer was confined to the area of perforation.

As already noted, the examination has not been technically difficult in spite of the fact that many of the patients are of the older age group and seriously ill. When performed carefully and without delay, the procedure is a safe and accurate means of demonstrating continued leakage from a perforated peptic ulcer, and the nature and location of the lesion. The demonstration of a normal stomach and duodenum aids in the prompt identification of those patients whose clinical picture simulates perforated peptic ulcer but who have some other condition (Fig. 9, *A* and *B*). It should be emphasized that, as in the 4 cases listed in Table v, there are those instances in which the ulcer crater is not demonstrated. However, there is no assurance that one could have shown these ulcers with a barium study. As a matter of fact, it is our impression that it is easier to demonstrate an ulcer crater, particularly of the duodenal bulb, with a water-soluble contrast medium than with barium.

SUMMARY

The roentgenographic determination of spontaneous sealing of perforated peptic ulcer has an important place in the nonoperative managment of this condition. A modified but thorough examination of the upper gastrointestinal tract using a water-soluble contrast medium was performed on 120 patients with suspected perforation.

Approximately 50 per cent of the proved perforated duodenal and benign gastric ulcers and all of the proved perforated malignant gastric ulcers were leaking at the time of examination. The leakage was generalized throughout the peritoneal cavity in almost one-half of these patients.

Leakage of contrast material was demonstrated in 6 cases (7 per cent) of perforated ulcer (4 duodenal, 1 benign gastric, 1 malignant gastric) in which there was no pneumoperitoneum.

The proportion of perforated ulcers which were sealed was not affected by age, sex, duration of symptoms of perforation, or chronicity of the ulcer.

By demonstrating a normal stomach and duodenum, the examination aided in the prompt identification of patients who had other conditions simulating perforated peptic ulcer.

The technique of examination is described. The examination is not difficult to perform. No patients have been harmed by the procedure.

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SIMPLE BENIGN PREPYLORIC ULCER*

THE POSSIBILITY OF AN UNEQUIVOCAL ROENTGEN DIAGNOSIS

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THE report of a benign prepyloric gastric ulcer on roentgen examination is accepted by the referring physician or consulting surgeon with considerable reservation because of skepticism that the radiologist can differentiate between benign and malignant ulceration in this part of the stomach. This belief apparently took firm hold in 1933 as a result of a report by Hampton[4] that all prepyloric ulcers observed at the Massachusetts General Hospital in a three year period were carcinomatous. This author therefore considered that the location of the ulceration was of overwhelming diagnostic importance, to the exclusion of all other features. In contrast, Singleton[13] in 1936 reported that in any given case of an ulcer within one inch of the pyloric sphincter, the probability was about four to one that it was benign. Doub[3] in 1940 studied 35 cases of ulceration of the pyloric and prepyloric areas believed to be benign or indeterminate on roentgen examination. These cases either failed to respond to medical management or were so extensive that a suspicion of carcinoma had to be entertained. Twenty-four turned out to be benign on pathologic study. Kirklin and MacCarthy[6] in 1942 reviewed all cases of ulceration of the prepyloric segment of the stomach operated upon at the Mayo Clinic from 1937 to 1941. Of a total of 132 cases, 63 were benign ulcers, 8 malignant ulcers and 61 ulcerated carcinomas. In 47 instances in which a niche was seen without evidence of tumefaction or other indication of malignancy, 24 cases were benign, 8 malignant and 5 were duodenal ulcers. These authors concluded that prepyloric ulcers were not more likely to be carcinomatous than ulcers situated elsewhere in the stomach. Collins[2] in 1952 analyzed 139 prepyloric lesions confirmed by surgery or follow-up. Of 54 cases said to be benign on roentgen examination, only 8 were proved to be malignant.

It is evident from the above brief and incomplete summary of the literature that the original report of Hampton has been contradicted many times. It is nevertheless surprisingly difficult to eradicate the impression originally made. It is not entirely clear why the report of Hampton is at such great variance with subsequent careful studies. One of the reasons may be that, as Hampton emphasized, he included only chronic cases of prepyloric ulceration that were associated with distinct induration. Since the emphasis in this article was on location of the lesion, the roentgen features were not described. In many of the reports which attempt to assess the accuracy of roentgen diagnosis in this area, evaluation is difficult because the effort made to demonstrate specific features and the degree of success in delineating them have necessarily varied a great deal. Moreover, from the roentgen point of view, the possibility of a correct differential diagnosis depends a great deal on the type of benign ulcer, *i.e.*, whether it is small and nonpenetrating or whether it is large and calloused. A penetrating ulcer completely destroys the muscularis propria layer and produces a large amount of granulation tissue and fibrous reaction which simulates roentgenologically, and often at exploration, a mass lesion. In contrast, a "simple" ulcer, that is, an ulceration which has not extended through the muscularis propria rarely presents as a pseudotumor unless it is relatively acute and associated with marked surrounding edema. In assessing the accuracy of roentgen diagnosis, it is essential to distinguish

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between these two types of benign ulceration. In the large majority of instances, it should be possible to recognize a simple ulcer as benign with practical certainty while differentiation between a penetrated or calloused benign ulcer and a small ulcerated carcinoma may be impossible roentgenologically. While there are unquestionably gradations between simple and penetrating ulcers, the distinction is nevertheless sufficiently clear-cut to be of considerable usefulness. The roentgen features or criteria of a benign ulcer, to be described below, may be used for all practical purposes as indicative of a simple chronic peptic ulcer.

The literature also contains conflicting reports[5,8] on the relative frequency of benign gastric ulceration in the various portions of the stomach. The results are likely to be different in resected specimens from those obtained at autopsy and both are likely to differ from series which include patients who do not come to operation or autopsy. The relative frequency of ulceration of the prepyloric portion of the stomach at autopsy[10] appears to be higher than in operative specimens. Moreover, Palmer[9] reported that small gastric ulcers, *i.e.*, ulcers not greater than 1 cm. in diameter, were most common in the pyloric and antral regions.

Over a period of years, we developed several clinical impressions about prepyloric benign ulcers—namely; (1) that they were considerably more common than much of the literature indicated; (2) that, in the large majority of instances, prepyloric ulcers were small and presented typical features of a benign ulcer; and (3) that when such features were present, the lesion could safely be called benign without reservation. Despite the fact that such cases were reported to be benign without equivocation, the possibility of carcinoma was usually permitted to sway clinical judgment. In several instances, resection was performed, despite the facts that the patient appeared to respond well to medical treatment and the pathologist had considerable difficulty in identifying any ulceration at all. These lesions had healed with little residue. In order to substantiate our clinical impressions, it was decided to review patients hospitalized over a five year period with benign ulceration of the prepyloric segment and with carcinoma in this area.

ROENTGEN CRITERIA

In conformity with others, the prepyloric segment of the stomach was considered to extend for an inch proximal to the pyloric sphincter. The concept of this segment as a separate unit is based on functional studies indicating that this segment acts as a distinct unit. Ulcerations located directly on the pyloric sphincter were not included as prepyloric ulcers since it was often uncertain whether these were duodenal or gastric.

Before presenting the results of the review of cases, it is necessary to indicate the roentgen features or the criteria which were used to arrive at the diagnosis of a benign prepyloric ulcer. Since these criteria were established after a preliminary review of the material, the fact that they fit the group of cases studied does not prove that they are necessarily the most suitable. Nevertheless, it was necessary to establish criteria in order to determine what fraction of the cases of benign ulceration in the prepyloric segment would thereby be included. Obviously, if the criteria were so stringent as to be useful in only a small percentage of cases, they would be of no great practical value. As demonstrated below, however, the criteria selected were fulfilled by all benign simple ulcers in this area. Moreover, they were not fulfilled in any case of carcinoma of the same region. It should not be assumed, however, that if an ulceration in the prepyloric portion of the stomach does not fulfill these particular criteria, it is necessarily malignant. There remains a group of lesions in which features of both benign ulceration and ulcerated carcinoma are present. Despite the fact that the weight of evidence may point in

Fig. 1. A small benign simple prepyloric ulcer is present along the lesser curvature with the typical appearance of a niche (arrow). A long neck is present, located in the center of a rather discrete ulcer mound. This pseudodefect is sharply demarcated and smooth in contour. A thick vertical fold extends inferiorly from the site of ulceration.

one or another direction in a particular case, the diagnosis must be considered to be indeterminate. In a third group of cases, the roentgen findings are unequivocal of carcinoma.

The criteria used to indicate that a simple benign prepyloric ulceration was present were the following: (1) A single discrete ulcer (Fig. 1) is clearly demonstrated within the prepyloric portion of the stomach. The major difficulty with this criterion is differentiation from duodenal ulcers at the base of the bulb. Cases were selected for study only if the position of the pylorus was clearly defined and the ulceration could be placed on the gastric side of the pyloric sphincter. (2) The ulcer is located along or near the lesser curvature. (3) The ulceration does not exceed 1 cm. in maximum diameter. (4) The mucosal pattern about the crater is intact (Fig. 2). In the case of ulcers that do not present as niches, radiation of folds to the edge of the crater is a necessary criterion (Fig. 3, *A–D*). When the ulcer presents as a niche, radiation of folds is sometimes not clear-cut but it must be evident that the fold pattern in immediate relationship to the crater is still present (Fig. 4, *A* and *B*). The folds usually appear somewhat thickened but the valleys between the folds are thin and sharply outlined. (5) Generalized spasm or persistent diffuse deformity of the prepyloric segment is absent. Cases in which the prepyloric segment was reduced to a narrow channel were excluded. This criterion can be phrased in another fashion —namely, that motor activity of the prepyloric segment in the nature of intermittent contraction is maintained. At some

Fig. 2. A simple prepyloric ulcer appears as a niche along the lesser curvature. A rather clear-cut radiating pattern of folds extends to the edge of the crater. The folds are thick but the valleys between the folds are relatively thin and sharply outlined. The gastric wall around the ulcer crater is flattened but there is no angulation or sharp demarcation from the adjacent normally distensible stomach wall. While the antrum never distended in completely normal fashion, the configuration of this portion of the stomach was not bizarre and intermittent contraction resembling normal motor activity of this portion of the stomach was present.

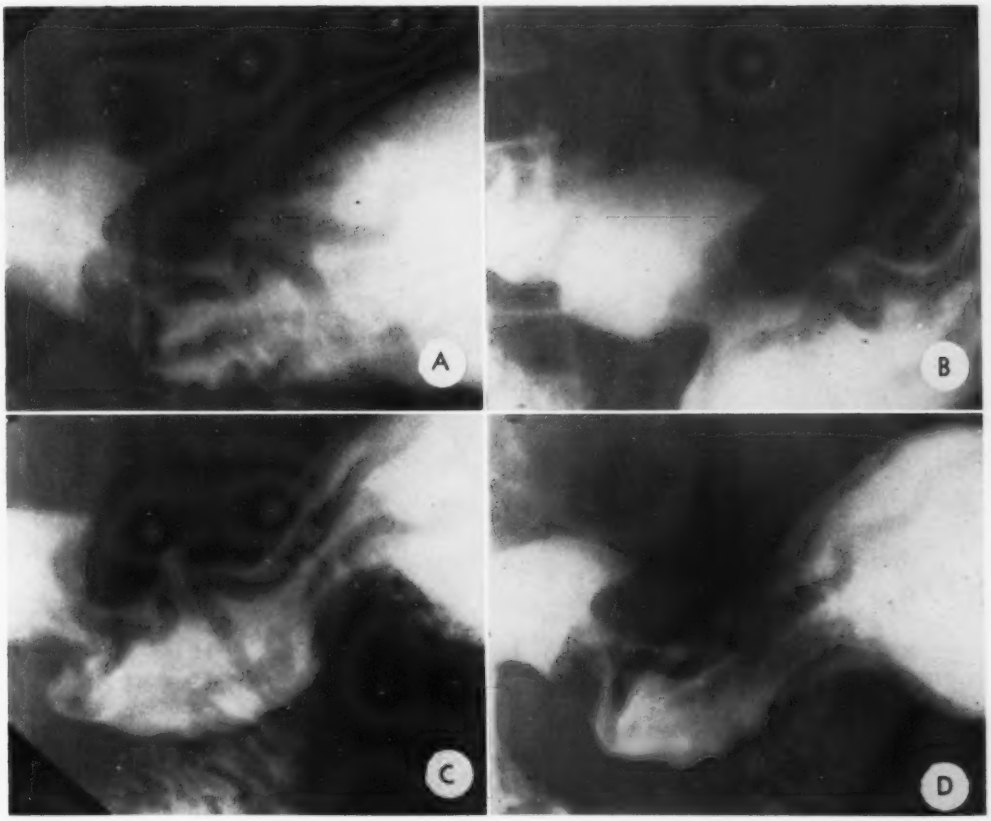

Fig. 3. (*A*, *B*, *C* and *D*). Serial roentgenograms on a polygraph of the prepyloric portion of the stomach showing a simple prepyloric ulcer. The patient is prone. The crater does not have the appearance of a niche. The varying fold pattern is well demonstrated. Exquisite radiation of the folds to the edge of the ulcer crater is evident only during certain phases of motor activity. The ulcerated area also participates in the motor activity of the region so that the crater is not always filled.

phase of filling or emptying of the prepyloric segment, the characteristic configuration of this portion of the stomach must be recognizable. (6) In only a rare case is there normal distensibility of the stomach wall about an ulcer crater. In practically every instance, there is an ulcer collar or mound[14] surrounding the crater which presents as a pseudofilling defect. For inclusion as a simple benign ulcer, it is required that this localized area of flattening or area of lack of distensibility be sharp and smooth in contour with the ulceration located in its central portion. Moreover, the ulceration associated with such an area of limited distensibility must have the typical appearance of a niche.[14] The "niche" appearance is good evidence that a rigid mass is not present (Fig. 5) since it indicates a saddle-shaped deformity rather than a hemispheric filling defect. This type of deformity is the basis of the "niche sign" and is the result of spasm of the muscularis mucosae and propria around the ulcer.

MATERIAL

The roentgen reports on all patients hospitalized in The Mount Sinai Hospital from 1953 to 1958 in whom a discharge diagnosis of gastric ulcer had been recorded were reviewed. An attempt was made to re-examine the roentgenograms of all cases in which the ulcer was located distal to the re-entrant angle. In the same fashion, all patients in whom the discharge diagnosis was carcinoma of the stomach

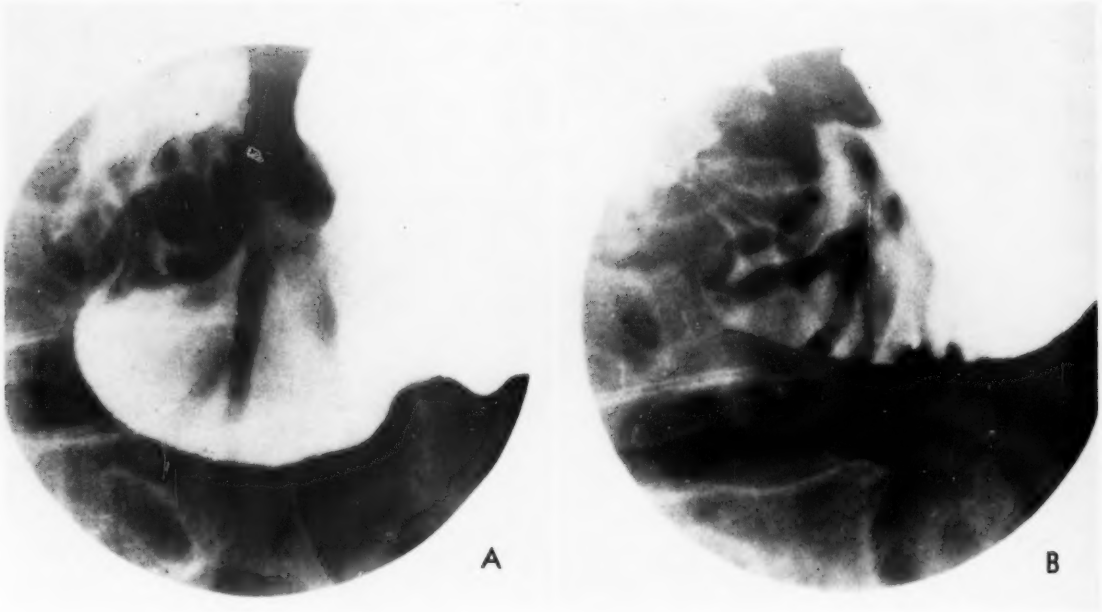

FIG. 4. (*A* and *B*) Serial spot roentgenograms with compression of the prepyloric area show a simple prepyloric ulcer with intact mucosal pattern. The mucosal folds were somewhat thickened and in the region of the crater were not completely effaced during distention. Note that the fold pattern is not stereotyped but changes in configuration presumably as a result of its own motor activity as well as contraction of the muscularis propria.

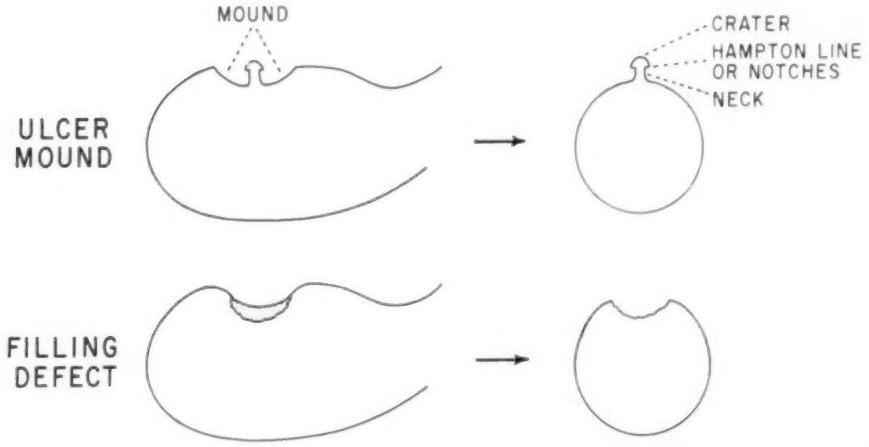

FIG. 5. Diagrammatic representation of the distinction between an ulcer mound and a filling defect. In the conventional frontal plane, both appear as defects in the contour. However, in cross section, a true filling defect is relatively symmetric while the lack of distensibility surrounding an ulcer crater is not evident as a defect. Spasm of the muscle around the crater creates a "saddle" type of deformity, convex in one plane and concave in another. For this reason, a benign crater in profile clearly projects from the contour as a niche. In contrast, a small filling defect has a variable appearance depending upon the degree of compression. An ulceration may be completely buried within it and evident only during compression. The profile features of a benign ulcer in addition to a neck with a soft-tissue collar around it include the Hampton line at the junction of the crater and the neck. A Hampton line is rarely seen in exquisite fashion with simple prepyloric ulceration but small notches at the same site can often be discerned.

were also reviewed and the roentgenograms in which the disease involved the distal part of the stomach were re-examined. The roentgen findings and the discharge diagnosis were correlated with available pathologic reports. In 12 instances, the microscopic slides were reviewed to confirm the fact that the lesions were truly chronic peptic ulcers and not erosions. All of these cases demonstrated that penetration of the ulceration extended at least through the muscularis mucosae and that the microscopic appearance of the tissue in the base of the ulcer was typically that of a peptic ulcer.

In the period of survey, there were 141 patients hospitalized and examined in the radiologic department who had a discharge diagnosis of benign gastric ulcer. In 51 of these cases, the distal portion of the stomach was involved but roentgenograms were available for re-study in only 45. Of these 45 cases, there was marked narrowing of the prepyloric segment in 4, in 6 cases differentiation from duodenal ulcer was uncertain, and in 3 cases the ulcers were so large as to extend beyond the prepyloric segment. In one case, a small ulcer was located proximal to the prepyloric segment but distal to the re-entrant angle. These 14 cases were therefore not included as examples of simple prepyloric peptic ulcer. Five additional cases were excluded from any further analysis because there was no adequate follow-up. All other cases were either confirmed by pathologic examination or during a course of follow-up which extended for at least twelve months. Twenty-six cases were therefore available for detailed study. In each case, review of the roentgenograms showed that the criteria for benign ulceration were fulfilled.

RESULTS OF ANALYSIS

The results indicate that in the hospital population, small benign ulcers in the prepyloric portion of the stomach constitute at least 25 per cent of the total number of gastric ulcers. The relative rarity of large ulcers in the prepyloric portion appears to be confirmed. Benign ulcers confined to the region between the prepyloric segment and the re-entrant angle are surprisingly uncommon.

During the same period, 160 hospitalized patients with carcinoma of the stomach were examined in the radiologic department. Eighty-four of these involved the distal portion of the stomach. In none were the criteria for simple benign prepyloric ulceration (Fig. 6, *A* and *B*) fulfilled.

The size of the ulcers on roentgen examination varied from 2 to 10 mm. The average and the median sizes were both 5 mm. Twenty-two of the ulcers were ovoid or elliptic, while 4 were somewhat rectangular. In 20 instances, the ulcers were visualized as niches projecting clearly from the lesser curvature. In 6 cases, the ulcers were seen only *en-face*. Table 1 indicates the location of the ulcers in relationship to the re-entrant angle and the pyloric ring. They appear to be rather uniformly distributed throughout the prepyloric segment. Definite radiation of folds to the ulcer crater was seen in 20 instances. In all cases, there was some enlargement or distortion of the mucosal fold pattern. In 2 cases, this was quite marked and associated with excessive secretions. In 5 instances, there was evidence of prolapse of gastric folds into the base of the duodenal bulb. In 2 instances, there appeared to be rather typical hypertrophy of the pyloric

TABLE I

LOCATION OF ULCERS

Distance from Pyloric Ring (cm.)	No. of Cases	Distance from Re-entrant Angle along Lesser Curvature (cm.)	No. of Cases
Just proximal	5	3.0	2
1.0	6	4.0	3
1.5	8	4.5	5
2.0	6	5.0	3
2.5	2	5.5	8
		6.0	5
		6.5	1

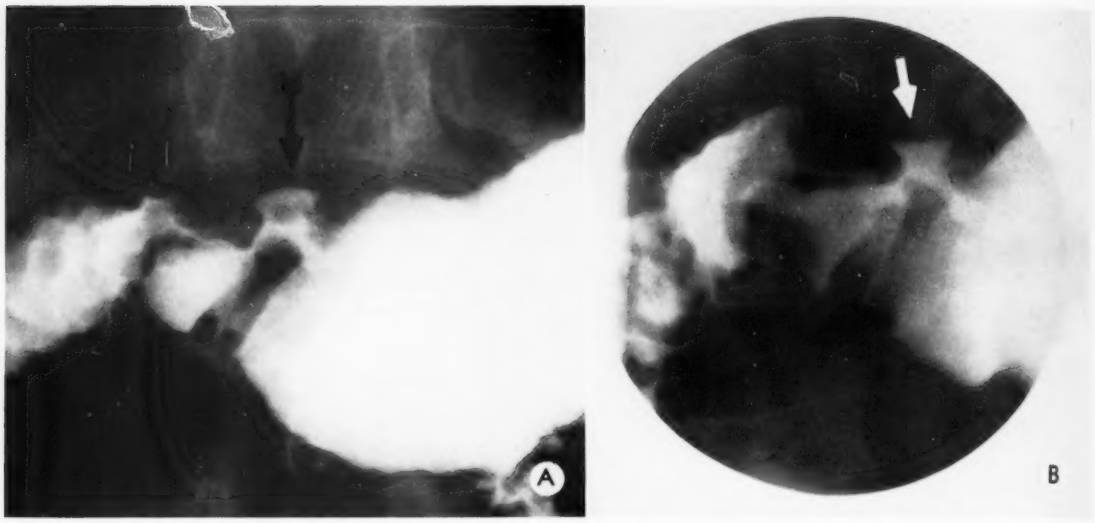

FIG. 6. A small ulcerating carcinoma of the prepyloric portion of the stomach. (*A*) A rather flat crater (arrow) is present along the lesser curvature in the center of a poorly demarcated defect. This roentgenogram shows the maximum amount of filling which was obtained in the prepyloric area throughout the course of the examination. The normal configuration of the stomach in this area was not seen at any time and typical motor activity was not present. (*B*) Spot roentgenogram with compression in the erect position shows the ulceration (arrow) with a fluid level. Several folds appear to radiate to the edge of the ulcer. However, a rolled edge which indents the crater is present along the lesser curvature at the distal margin of the ulceration. The folds distally along the lesser curvature stop abruptly at the margin of this rolled edge. A persistent bizarre configuration of the prepyloric segment plus evidence of a rolled edge along one border violate the criteria of a simple benign ulcer. This case was selected for illustration because, in the series studied, it came closer than any other to fulfilling the criteria described in the text for simple benign ulceration.

musculature. In one of these cases, confirmation was obtained at operation. In 5 instances, there was deformity of the duodenal bulb characteristic of a duodenal ulcer. There was one case with a second gastric ulcer on the posterior wall of the body of the stomach.

CLINICAL FEATURES

The clinical features in the group of 26 patients were analyzed within the limitations of the information available. The age of these patients varied between thirty-nine and eighty-three. The average and the median ages were both fifty-seven. There were 15 females and 11 males. Twenty-four patients were white and 2 were Negroes. In 2 cases, ulceration was associated with steroid therapy. Epigastric pain was present in 23 of these patients. In 6 patients, pain had been present intermittently over a period of more than five years. In 8 patients, pain had been present from one to five years. In 17 instances there was a clear history of relief by food. Frequent vomiting was a symptom in 6 instances. Weight loss, however, was a prominent feature in only 2 patients. Gross gastrointestinal bleeding was noted in 12 cases, while anemia with positive guaiac stools was present in 2 patients. One case developed a free perforation. Free acid was present in 16 cases, *i.e.*, in all cases in which the test was recorded. Pyloric obstruction was not a prominent feature in any case. In general, the symptoms did not appear to vary significantly from those of a duodenal ulcer. The fact that bleeding was a frequent feature is to be expected since this series included only hospitalized patients. It is rather surprising, however, that massive hematemesis

may result from ulcerations which have not penetrated into the serosa.

FOLLOW-UP ROENTGEN STUDY

In 8 cases, follow-up roentgenograms were available. In 2 patients, an ulcer was still present, somewhat smaller in one and unchanged in the other, seventeen and twenty-seven months respectively following the original examination. A tiny niche-like projection may persist after healing because of wrinkled folds overlying the scarred area which prevent complete distention and stretching of the mucosa (Fig. 7, *A* and *B*). In no case was there complete restoration of the prepyloric segment to an absolutely normal appearance. In some, there was only a hardly noticeable flattening or lack of distensibility of a small region of the lesser curvature, while in others a thick transverse fold or incisura-like defect remained (Fig. 8, *A* and *B*, and 9, *A* and *B*). In several cases, the persistent lack of distensibility over a short segment associated with a flattened or distorted mucosal pattern created a picture which was more difficult to differentiate from carcinoma than the original lesion (Fig. 10, *A* and *B*). It is likely that many benign antral deformities are the result of a healed prepyloric ulcer. While no cases of recurrent ulceration were noted in this group of patients, we have seen several instances of healing of a prepyloric ulcer followed by recurrence in the same area sometimes on repeated occasions. This sequence of events is quite comparable to that seen so frequently in patients with duodenal ulcers.

DISCUSSION

The roentgen features of benign ulceration and of carcinoma in the prepyloric segment of the stomach do not differ essentially from those in other areas of the stom-

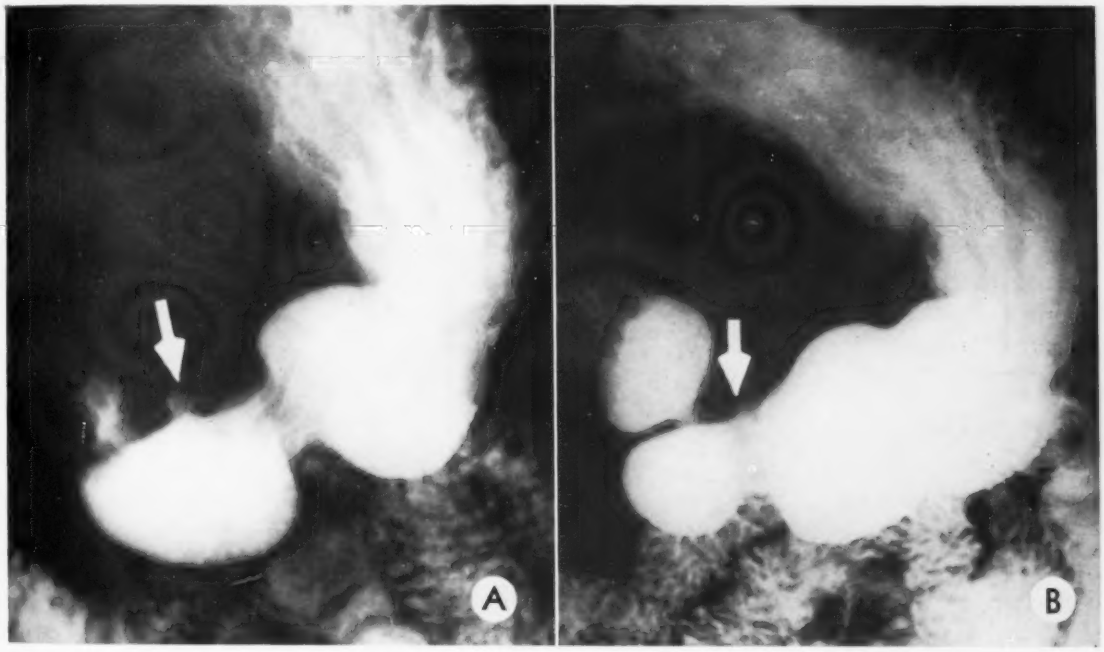

Fig. 7. (*A*) A simple prepyloric ulcer (arrow) is present along the lesser curvature which presents as a niche in the center of a flattened area. Intact radiating folds can be faintly seen extending to the edge of the ulcer. (*B*) Roentgenogram five months later shows a tiny niche-like projection (arrow) at the site of the ulceration. At this stage, it is difficult to distinguish between barium caught between distorted folds and residual ulceration. The folds adjacent to a healed scarred area persist despite maximum distention. A wrinkling of the contour with a slight diminution in the density of the barium in the area results.

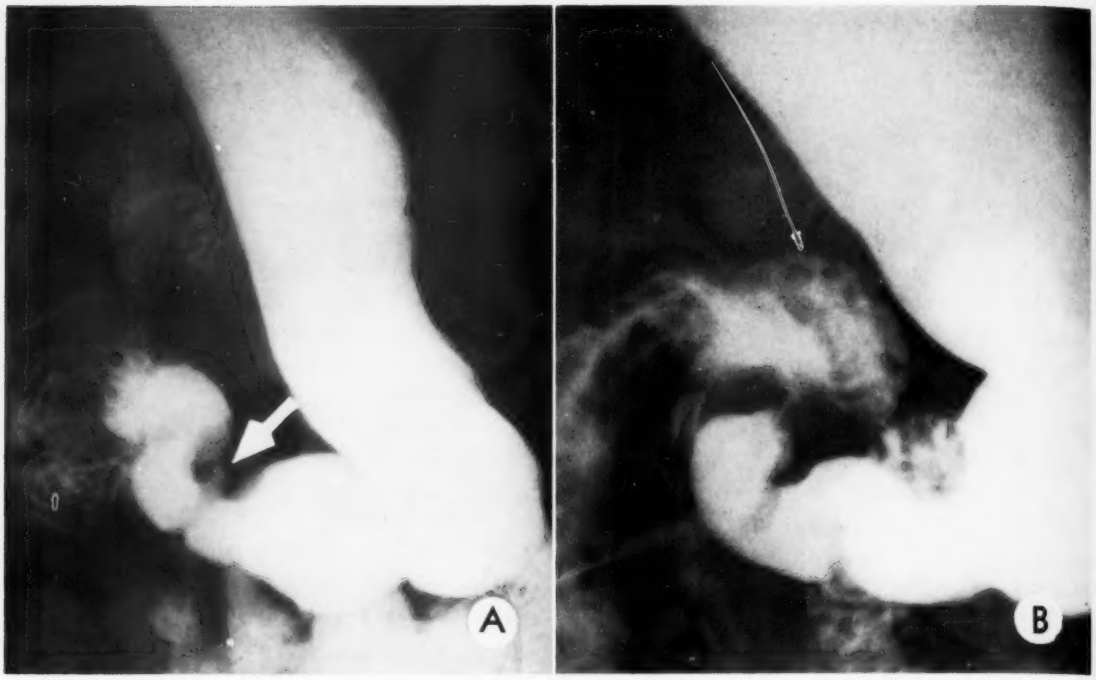

FIG. 8. (*A*) A simple prepyloric ulcer shows a typical niche appearance (arrow) within a rather discrete ulcer mound. A Hampton line is faintly seen. A thick vertical fold connects this area with the greater curvature. (*B*) Roentgenogram four months later shows no definite evidence of an ulcer crater but an incisura-like defect persists at the site of previous ulceration. The thick vertical fold is still present and is not effaced during maximum distention. The lesser curvature is shortened at the site of the incisura, as evidenced by the turning up of the stomach distal to this point.

ach.[1,12] This segment has received special attention only because it has been considered that the conventional criteria are misleading in this region. The distal portion of the stomach is often easier to examine than the remainder of the stomach because of its smaller size and the ability to apply graded compression more conveniently. Moreover, the mucosal pattern in this area of the stomach is ordinarily simple to demonstrate and constant in configuration, and motor activity is more pronounced than elsewhere in the stomach. Small benign ulcers, a few millimeters in diameter, appear to be unusually frequent in this area as compared with the remainder of the stomach, but this may be due to limitations of the method of examination in other areas. It is true that, more commonly than elsewhere in the stomach, benign ulceration of the prepyloric portion of the stomach may be associated with marked spasm and perhaps gastritis. The roentgen features of the ulceration *per se* may therefore be more difficult to delineate. Similar spasm may be produced by a malignant ulcer or an ulcerated carcinoma or an infiltrating scirrhous carcinoma. As pointed out above, when diffuse or bizarre narrowing is present, an unequivocal diagnosis of a simple benign prepyloric ulcer cannot be made.

It is becoming general practice for the radiologist to report ulcerated lesions of the stomach as benign, indeterminate or malignant.[10,11,12] The indeterminate group actually consists of two subgroups; namely, one in which the examination is unsatisfactory because of excessive secretions or bizarre motor activity and a second in which the roentgen features do not all point in the same direction. This is of considerable assistance in evaluating the accuracy of roentgen reports.

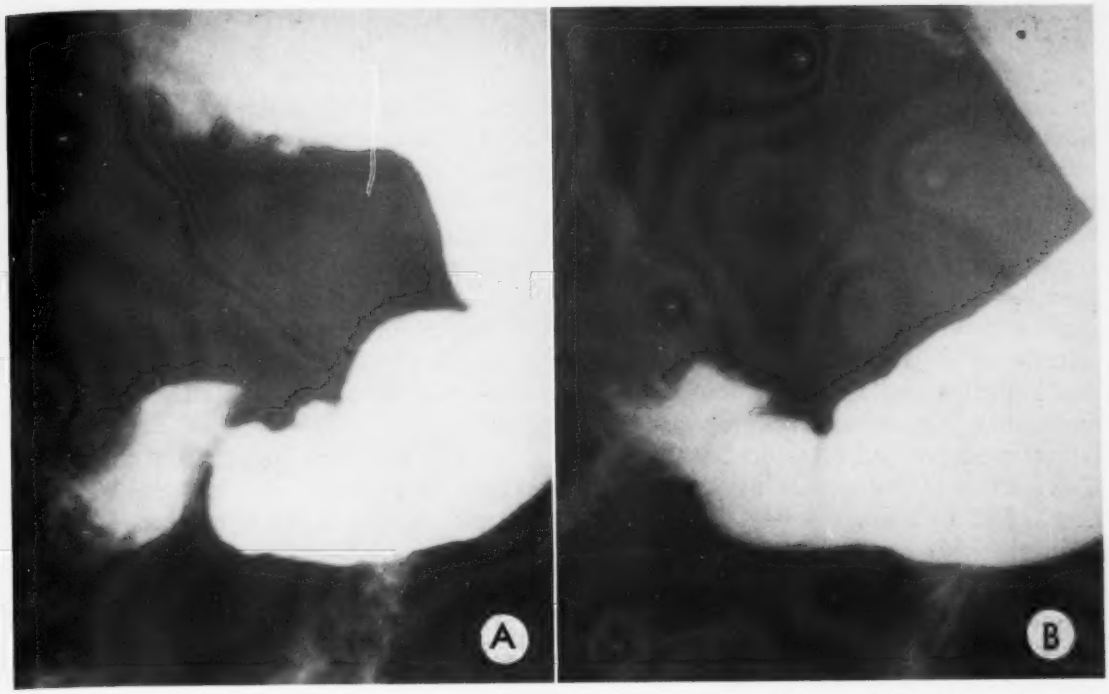

Fig. 9. (*A*) A simple prepyloric ulcer is present along the lesser curvature with an atypical niche appearance. There is no neck to the crater. (*B*) Roentgenogram one year later shows no evidence of ulceration but a small incisura or a thick fold is present at the same site.

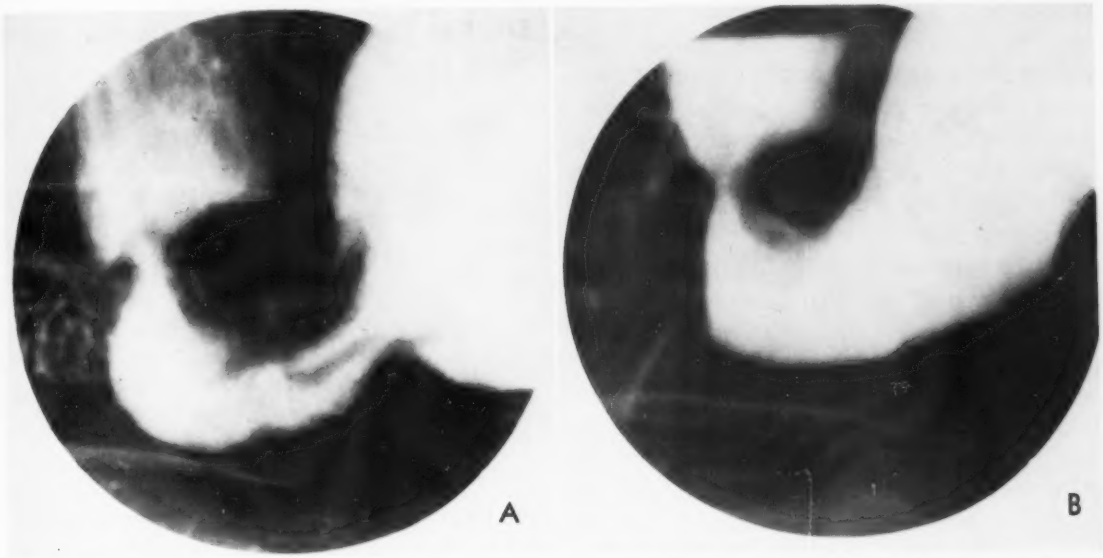

Fig. 10. (*A*) A small prepyloric ulcer is present in the center of an ulcer mound. The fold pattern is intact and extends to the edge of the crater although radiation of folds is not evident. (*B*) Roentgenogram four months later shows that the ulceration has healed but there is a localized area of decreased distensibility. On compression studies, however, the fold pattern in this area appeared to be intact and the configuration changed during different phases of filling. The patient has remained well for a period of four years.

The results of the study described above may be legitimately criticized on the basis that it was started with preconceived ideas and conducted with personal bias. It must also be admitted that the results may be coincidental to some extent since it is likely that few small malignant ulcers will be represented in any series of symptomatic patients. There is no question that, with additional experience, a malignant prepyloric ulcer will be found which fulfills all of the criteria of a simple benign ulcer roentgenologically and on gross pathologic examination. Nevertheless, the practical fact is that we have had no difficulty clinically. Carcinomas of the stomach have not been overlooked by using the criteria described. This statement applies not only to the hospitalized series of cases studied in this report but to a considerably larger group of patients seen and followed on an ambulatory basis by ourselves and our colleagues. Another criterion of the benign nature of a gastric ulcer in the prepyloric segment of the stomach or elsewhere, however, should be added. This is essentially a clinical criterion; *i.e.*, a test of treatment. In every case, it is recommended that close supervision and intensive medical care be practiced until the ulcer is completely healed.[7] A recommendation for re-examination after a stated interval of time is included in the radiologist's report. One cannot, however, insist that the roentgen appearance of the stomach return completely to normal, since some deformity usually persists. It should be possible to demonstrate that the abnormality is simply a deformity; that is, no defect or rigid area remains and an intact fold pattern is present. In any particular case, the decision for or against operative intervention should be made in a fashion similar to that with duodenal ulcer, on the basis of the clinical course of the patient rather than the fear of overlooking a carcinoma.

SUMMARY

1. It is not true that the majority of prepyloric ulcers are malignant. On the contrary, simple ulcers of this area are overwhelmingly benign.

2. From the point of view of the roentgen differential diagnosis of prepyloric ulcers, it is important to distinguish between simple, nonpenetrating ulcers and penetrating calloused ulcers. A correct roentgen diagnosis is much simpler in the first group.

3. The roentgen criteria of simple peptic ulceration of the prepyloric portion of the stomach are adequate to exclude the possibility of carcinoma for all practical purposes.

4. The roentgen findings and the clinical course of patients with simple prepyloric gastric ulceration are not greatly different from those of patients with duodenal ulcer. It is common for a deformity to persist after healing of a benign gastric ulcer.

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THE ROENTGEN APPEARANCE OF SPONTANEOUS INTRAMURAL HEMORRHAGE OF THE SMALL INTESTINE ASSOCIATED WITH ANTI-COAGULANT THERAPY*

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THE use of anticoagulant therapy in the management of diseases associated with occlusive vascular changes has occasionally given rise to complications due to the defective clotting mechanism. Since patients with some of these diseases, such as cerebral or coronary thrombosis, are poor surgical risks, it is imperative to recognize that the clinical findings suggesting an acute surgical abdomen may be secondary to intramural or intramesenteric bleeding of the intestine, a condition often lending itself to nonoperative clinical management. Spencer, Bateman and Horn,[6] in their review of 23 cases of intramural hematoma which caused small intestinal obstruction, observed that in more than two-thirds of the cases there was a history of antecedent trauma. In 2 cases, a definite bleeding tendency was observed; one a hemophiliac, the other a newborn infant with an undiagnosed hemorrhagic diathesis. No mention was made of the association of anticoagulant therapy with any of the reported cases. The first report of spontaneous intramural hemorrhage of the small bowel associated with anticoagulant therapy was made by Berman and Mainella[1] in 1952. They reported 5 cases presenting with overt hemorrhage. Of these, 3 had findings of an acute surgical abdomen. Mendelsohn and Iglauer[4] reported 2 cases of spontaneous intraperitoneal bleeding due to dicumarol intoxication. Single cases of spontaneous localized intramural hemorrhage with surgical resection of the involved small intestinal segment occurring in patients under anticoagulant therapy were described by Pearson and MacKenzie[5] and by Gilbert and Jorgenson.[3]

No contrast studies of the small intestine with intramural hemorrhage resulting from anticoagulant therapy have been reported. The 3 cases to be described in this report were studied with barium and a consistent and characteristic roentgen appearance was demonstrated. The roentgen findings in the small intestinal study in the first case were correlated with the anatomic changes found during surgery and were sufficiently typical to suggest the correct roentgenologic diagnosis in the 2 subsequent cases. Characteristically, the findings are those of a rigid segment of small intestine with an intact coarsened mucosal pattern. The lumen is diminished and narrow spike-like projections of barium extend to outline the normal width of the bowel. The appearance mimics an infiltrative process of the bowel wall.

REPORT OF CASES

CASE 1. A sixty year old white male was admitted to the Jewish Hospital on September 10, 1957, with the chief complaint of diffuse abdominal pain of two days' duration. Three months earlier, he had been hospitalized because of left sided headaches, right homonymous hemianopsia, and right hypesthesia secondary to a carotid artery occlusion. Dicumarol therapy was initiated and maintained after the patient was discharged from the hospital. Prothrombin times were checked weekly, but three days prior to this admission the patient failed to keep his laboratory appointment. Two days before admission the patient complained of low midabdominal pain which then became generalized and was accompanied by nausea and vomiting. The symptoms became progressively more severe. He had had no bowel movements for the twenty-four hours preceding hospitalization. The physical examination

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showed a well developed, well nourished male in acute abdominal distress, lying supine with his legs drawn up. The blood pressure was 160/100, the pulse rate 100 and regular, and the temperature 100° F. The skin showed no ecchymoses. The abnormal physical findings were limited to the abdomen. There was no distention. The bowel sounds were absent. There was diffuse muscle guard, more marked on the right. Tenderness to rebound was present throughout. There was right costovertebral angle tenderness. Laboratory examination revealed a hemoglobin of 14.7 gm. per cent and a white blood cell count of 12,900. The prothrombin time was 5 per cent of normal, and the serum diastase was 120 Somogyi units. Urinalysis, including microscopic examination of the sediment, was negative.

The patient was thought to have an acute surgical abdomen, but operation was postponed because of the low prothrombin time. The patient was given 50 mg. of mephyton intravenously. A Levine tube was passed and connected to continuous suction and wide spectrum antibiotics were started. Five hours later, the hemoglobin had dropped to 11.4 gm. per cent and the prothrombin time had increased to 36 per cent of normal. A fleck of stool on the rectal examining finger was hemotest positive. On the second hospital day, the abdomen continued to be rigid and diffusely tender and the temperature rose to 102° F. The white blood cell count was 13,500 and the prothrombin time had risen to 70 per cent. The patient began to improve on the third day, showing less rigidity and some return of bowel sounds, but continued to show diffuse abdominal tenderness. The patient was transfused with one unit of blood. The white blood cell count had dropped to 8,000. The patient continued to improve, and on the fourth day the Levine tube was removed. On the fifth day, the patient passed two tarry stools. Antibiotics were discontinued on the sixth day, and the patient became afebrile. The tenderness subsided on the seventh day, and the patient was now asymptomatic. On the tenth hospital day, a barium study of the small intestine showed a segment of proximal jejunum approximately 30 cm. in length with a narrowed lumen, rigid wall and coarsened edematous valvulae conniventes (Fig. 1). Multiple diverticula and atypical spike-like projections of barium outlining the normal caliber of the bowel lumen were present. Because of the findings of diverticula together with an apparent infiltrating lesion of the bowel wall, a diagnosis of diverticulitis of the jejunum was made and surgery was advised. At operation, a moderate amount of old blood was present in the peritoneal cavity. A segment of the small intestine (Fig. 2) about one foot distal to the ligament of Treitz showed a number of very large diverticula and diffuse hemorrhage into the bowel wall. This segment of small intestine was resected. Microscopic examination showed the mucosa to be intact (Fig. 3). Within the submucosa, there was widespread hemorrhage with no cellular reaction. There was moderate edema and hemorrhage throughout all layers. The pathologist's diagnosis was intramural hemorrhage of the small bowel with localized jejunal diverticulosis.

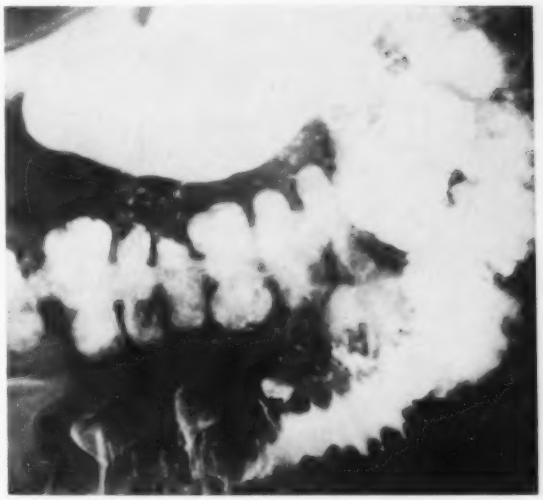

Fig. 1. Case 1. Examination of the small intestine one hour after the barium meal shows in a segment of the proximal jejunum (arrows) the characteristic roentgen appearance of intact mucosa, coarsened edematous folds and spike-like projections extending out to the normal luminal caliber. Note the presence of diverticula in this segment. The barium in the colon is from a previous barium enema study.

Comment. The presence of diverticulosis of the jejunum was a misleading factor, and the striking serrated appearance of the involved segment of the small intestine in an area of extensive diverticulosis led to an incorrect diagnosis of diverticulitis of the jejunum. Even though the patient had im-

FIG. 2. Case I. A photograph of the surgical specimen showing the sharply demarcated hemorrhagic segment.

proved markedly on a conservative regimen, surgery was decided upon because of the bizarre roentgen appearance in the jejunum. It is interesting to speculate that the presence of diverticula in the jejunum may have contributed to the localization of the massive hemorrhage to this portion of the small intestine.

CASE II. A sixty-seven year old Negro female entered the Jewish Hospital for the second time on June 8, 1959, complaining of fever and abdominal pain. Four months earlier, the patient was admitted to the hospital because of myocardial ischemia. She was started on dicumarol therapy which was continued after discharge. Five days prior to this admission, the patient began having chills, fever and abdominal pain. This was soon followed by abdominal distention, nausea and vomiting. The prothrombin time was at a therapeutic level. Subsequently, the temperature rose to 103° F., and the distention increased. She became progressively worse, and one day prior to admission she was unable to retain anything by mouth. The patient gave the history of having had an appendectomy thirty years previously. On physical examination, the blood pressure was 120/68, the pulse rate was 120 and regular, and the temperature was 101° F. The skin showed no hemorrhages or petechiae. The positive physical findings were limited to the abdomen. There was distention and the bowel sounds were hypoactive. The entire abdomen was tympanitic to percussion except in the flanks. There was generalized tenderness with moderate rebound tenderness throughout. There was a vague right upper quadrant mass. The laboratory examination revealed a hemoglobin of 8.8 gm. per cent and a white blood

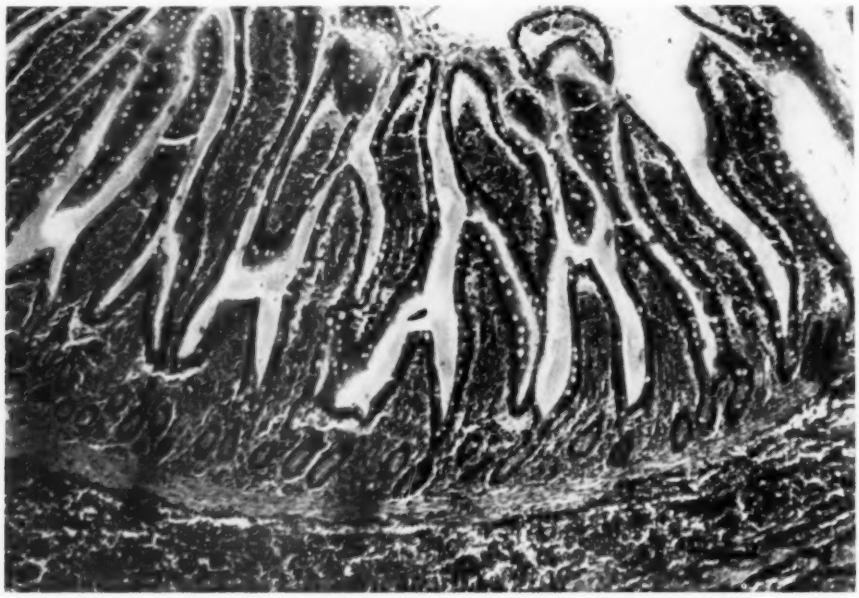

FIG. 3. Case I. A photomicrograph of the wall of the resected bowel showing an intact normal appearing mucosa and diffuse submucosal hemorrhage.

cell count of 17,600 with 33 per cent stabs. The urinalysis was negative. Roentgen examination of the abdomen revealed gas in numerous loops of small bowel and also throughout the colon. A diagnosis of adynamic ileus was made. On the second hospital day, the patient's temperature rose to 103° F. Abdominal distention continued with hypoactive bowel sounds. The hemoglobin dropped to 6.8, then to 6.0 gm. per cent. The white blood cell count rose to 19,100, and then to 21,300 with 48 per cent stabs. The prothrombin time was markedly prolonged. Fifty milligrams of mephyton were given intravenously, and two units of blood were administered. A barium enema examination showed a normal colon without evidence of obstruction. Because the postevacuation roentgenograms showed complete emptying of the large intestine, the intestinal tonus was thought to be normal. Due to the presence of continued distention of the small intestinal loops, the diagnosis was changed to incomplete mechanical small intestinal obstruction. A barium study of the small intestine (Fig. 4) showed a narrowed rigid segment of proximal jejunum. There was considerable coarsening of the valvulae conniventes. Sharp spike-like projections of barium extended between coarsened mucosal folds to outline a lumen of normal size. The small intestine distal to this area was distended. A diagnosis of intramural hemorrhage of the proximal jejunum was made as well as small intestinal obstruction at a more distal point. Since the hemoglobin had risen only to 8.2 gm. per cent, two more units of blood were administered. The white blood cell count remained at 20,400. On the fourth hospital day the temperature rose to 104° F., the hemoglobin to 12.5 gm. per cent and the prothrombin time was 90 per cent of normal. Because there was no improvement in the patient's clinical condition, a laparotomy was performed. At operation, a perforated appendix with localized abscess formation was found and removed. A 15 cm. segment of the jejunum starting at the ligament of Treitz was thickened and deeply hemorrhagic. The mesentery was also hemorrhagic. Approximately 18 inches distal to this segment there was another hemorrhagic segment, not as severely involved as the first, which was resected. On cut section, the wall of the specimen was markedly thickened with hemorrhage in all layers of the intestine and in the mesentery. Grossly, the mucosa appeared intact. Microscopic examination revealed hemorrhage permeating all the layers of the bowel dissecting an intact mucosa from the submucosa. The appendix was acutely inflamed and perforated. On the first postoperative day the patient became afebrile and, except for a wound infection, made an uneventful recovery. Nineteen days later a repeat barium study of the small intestine showed the proximal jejunum to be normal (Fig. 5). The lesion previously demonstrated on roentgenograms and proved at surgery had shown complete anatomic reversibility. Another examination six months later again revealed no jejunal abnormality.

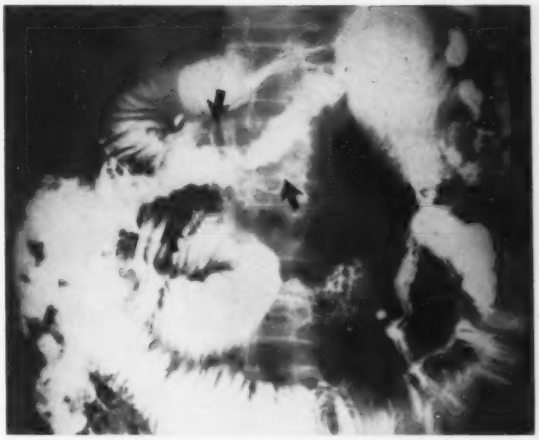

Fig. 4. Case 11. Roentgenogram of the gastrointestinal tract at one hour after the barium meal showing the long rigid infiltrated appearing segment of the most proximal jejunum (arrows). The small intestine is greatly distended, indicative of an obstruction at a more distal point.

Comment. In this case an acute appendicitis with perforation and localized abscess formation was complicated by a spontaneous intramural hemorrhage into two areas of the jejunum. It is possible that peritonitis secondary to the perforated appendix may have been a factor in producing a rather rapid lowering of an otherwise well controlled prothrombin level. Roentgenograms of the small intestine demonstrated extensive changes of the proximal jejunum secondary to intramural hemorrhage. This was confirmed at operation. Two follow-up roentgen examinations, made nineteen days

FIG. 5. Case II. Repeat barium study made nineteen days after Figure 4 shows a completely normal small intestinal pattern.

and again six months later, showed complete anatomic reversibility of the lesion.

CASE III. A forty-five year old white male was admitted to the Jewish Hospital for the third time on June 3, 1960, complaining of a stomach ache and bruises. Five months earlier, he was admitted to the hospital for the first time with a diagnosis of acute myocardial infarction. He was hospitalized for four weeks and started on dicumarol which was continued after discharge. One month prior to the present admission, he was hospitalized for the second time because of retrosternal pain, and was discharged six days later with a diagnosis of coronary insufficiency. The dicumarol therapy was continued throughout that admission and after discharge. Three days prior to this admission, the patient experienced the onset of nausea, aching localized in the muscles of his legs, as well as generalized myalgia. For the previous twenty-four hours he noted multiple skin ecchymoses and the onset of bilateral lower quadrant abdominal aching with increasing generalized myalgia. On physical examination, the blood pressure was 130/70, the pulse rate was 96 and regular, and the temperature was 99° F. The patient was a well developed, well nourished male with multiple ecchymoses and subcutaneous hematomas. The abdomen was slightly protuberant, and the bowel sounds were hypoactive. There was no muscle guard. There was moderate generalized tenderness and rebound tenderness more marked in the lower abdomen. Laboratory examination revealed a hemoglobin of 13.8 gm. per cent. The white blood cell count was 16,150 with 5 per cent stabs. The prothrombin time was 12.8 per cent of normal. The stool was faintly positive for occult blood. Roentgenograms of the abdomen showed several nondistended gas-containing loops of small intestine with gas and feces present in the colon. This was interpreted as indicative of an adynamic ileus. A small intestinal series was requested because of the possibility of a spontaneous intramural hemorrhage. The patient was given nothing by mouth and was administered 50 mg. of mephyton intravenously. On the evening of admission the temperature was 100.8° F.

The next day the patient's condition improved. The bowel sounds were active and now there was only minimal lower abdominal tenderness. The patient was afebrile, and the white blood cell count had dropped to 8,500. The prothrombin time was now normal. The stool was positive for occult blood. The patient continued to improve and on the fourth hospital day a barium study of the small intestine was obtained. A segment of the midileum measuring approximately 25 cm. in length showed narrowing of the lumen, coarsening of an intact mucosa and some spike-like projections of barium outlining the normal caliber of the lumen (Fig. 6, *A* and *B*). A roentgen diagnosis of spontaneous intramural hemorrhage of the small intestine was made. The patient's condition continued to improve, and he was discharged one week after admission. Two weeks after discharge, a repeat small intestinal series revealed no abnormality in the bowel pattern.

Comment. This patient, on anticoagulant therapy because of coronary occlusive disease, developed symptoms and signs of an acute surgical abdomen which responded to conservative treatment. Roentgen examination showed the characteristic changes of spontaneous intramural hemorrhage in the midileum. Complete reversibility of the process was demonstrated on roentgenograms made three weeks after the acute episode.

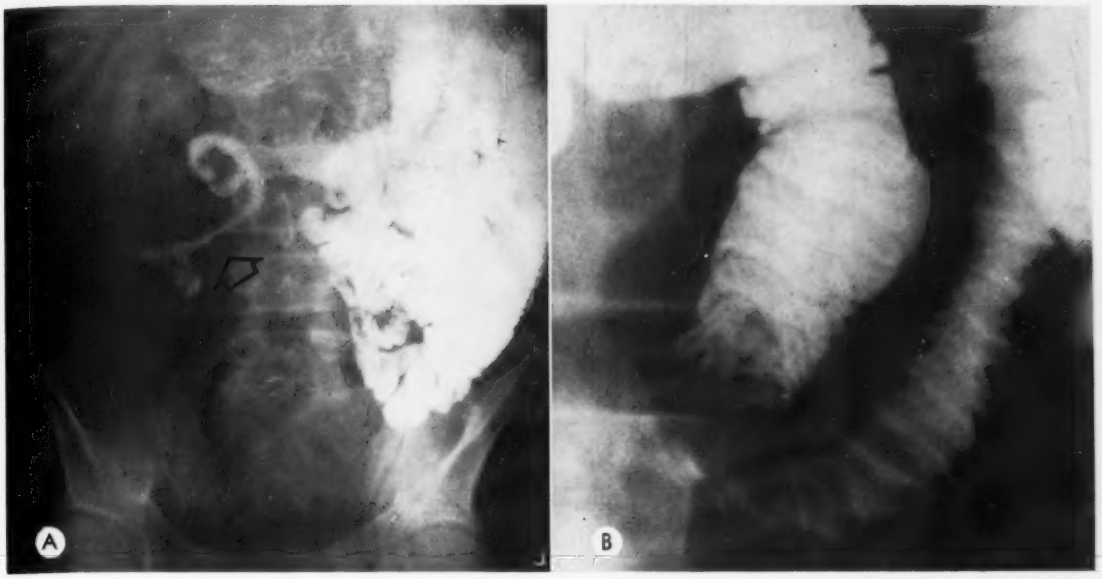

Fig. 6. Case III. (*A*) Barium study of the small intestine shows delay in intestinal motility in the distal ileum indicative of incomplete intestinal obstruction. (*B*) Detail spot roentgenogram of the abnormal ileal segment demonstrates the characteristic findings of spontaneous intramural hemorrhage.

DISCUSSION

There are many reports in the surgical and radiologic literature of intramural hematoma of the small intestine. Most of these occurred in the duodenum and were secondary to trauma. Felson and Levin[2] described the characteristic roentgen findings of traumatic intramural hematoma. They considered the presence of an intramural tumor mass, a coiled spring appearance and coarsening of the mucosal folds in the adjacent intestine pathognomonic of intramural hematoma. This paper summarizes the clinical and roentgen findings of spontaneous intramural hemorrhage in the small intestine occurring during anticoagulant therapy. The important clinical and laboratory findings from 6 of the previously recorded cases and the 3 cases reported here are presented in Table 1. These data suggest the following typical case history.

A patient on anticoagulant therapy because of occlusive vascular disease suddenly develops abdominal pain, usually cramping in nature, accompanied by nausea and vomiting and low grade fever. Physical examination of the abdomen reveals it to be distended with the bowel sounds hypoactive to absent. There is tenderness to abdominal palpation, both direct and rebound. Muscle guard may or may not be present. A vague mass may be palpable. Gross bleeding into the skin or from the gastrointestinal or urinary tract may make the hemorrhagic state obvious. A drop in hemoglobin or laboratory testing of the stool and urine may show evidence of occult blood loss. The prothrombin time is below therapeutic levels. The white blood cell count is elevated. Roentgenograms of the abdomen reveal numerous gas-distended loops of small intestine, but with gas also visualized along the course of the colon. Barium studies of the small intestine demonstrate the characteristic findings of spontaneous intramural hemorrhage. These are well demonstrated on the roentgenogram of the resected small intestinal segment from our Case II after distention with barium (Fig. 7). The appearance is that of an infiltrative process of the intestinal wall rather than that of a localized intramural tumor. The segment of small intestine is narrowed and rigid and

TABLE I

SYMPTOMS, CLINICAL FINDINGS AND LABORATORY DATA IN 9 CASES OF SPONTANEOUS INTRAMURAL HEMORRHAGE OF THE SMALL INTESTINE

		Symptoms		Findings on Physical Examination of Abdomen						Laboratory Findings						
													Evidence of Abnormal Bleeding			
													Gross		Occult	
No.	Authors	Abdominal Pain	Nausea and Vomiting	Distention	Bowel Sounds	Muscle Guard	Tenderness	Rebound Tenderness	Temperature (F)	White Blood Cell Count	Prothrombin Time	Drop in Hb	Gastrointestinal Tract	Urinary Tract	Stool	Urine
1	Berman and Mainella[1]	present (cramping)	present	present			present		102.0°			present	present		present	
2	Berman and Mainella[1]	present (cramping)	present	present			present		101.0°		30 sec.					
3	Mendelsohn and Iglauer[4]	present (cramping)	present	present	absent		present				8%			present		present
4	Mendelsohn and Iglauer[4]	present	present	present		present	present	present	99.8°	12,600	0%					present
5	Gilbert and Jorgenson[3]	present (cramping)	absent (anorexia)	absent	hyperactive		present		99.7°	8,600	11%	present			absent	present
6	Pearson and MacKenzie[5]	present (cramping)		present	absent				98.6°		L.10%		present		present	absent
7	Senturia *et al.*	present		absent	absent	present	present	present	102.0°	13,500	5%	present	present	absent	present	absent
8	Senturia *et al.**	present	present	present	hypoactive	present	present		104.0°	21,300	prolonged	present	absent	absent	none reported	
9	Senturia *et al.*	present	present	present	hypoactive	absent	present	present	99.0°	16,150	12.8%		absent	absent	present	absent

* This case (our Case 11) was complicated by the presence of acute appendicitis with perforation and abscess formation which distorted the clinical picture.

shows a coarsened, intact mucosal pattern. There are occasional narrow spike-like projections of barium extending to outline the normal width of the lumen. The segment is rigid because of hemorrhage permeating through all layers of the intestine. There is narrowing of the lumen because the relatively nondistensible serosa causes the hemorrhage to expand centripetally, and therefore, encroach upon the lumen. The intact mucosa is rigid and coarsened in appearance because of the dissection of hemorrhage into the easily separable plane between the muscularis mucosae and the muscular layer.

In 2 of our cases, we have roentgenographically observed this lesion to be completely reversible and amenable to nonsurgical management. It is, therefore, imperative to recognize this condition in patients on anticoagulant therapy who may be poor operative risks due to severe coronary or cerebrovascular disease.

SUMMARY

1. Three cases of spontaneous intramural hemorrhage of the small intestine occurring in patients on anticoagulant therapy are reported.

2. The clinical findings in these cases and in 6 of the collected cases from the literature are presented.

3. A characteristic roentgen finding on barium study of the small intestine in these cases is described.

4. Serial roentgenograms show complete anatomic reversibility of these abnormal small bowel changes.

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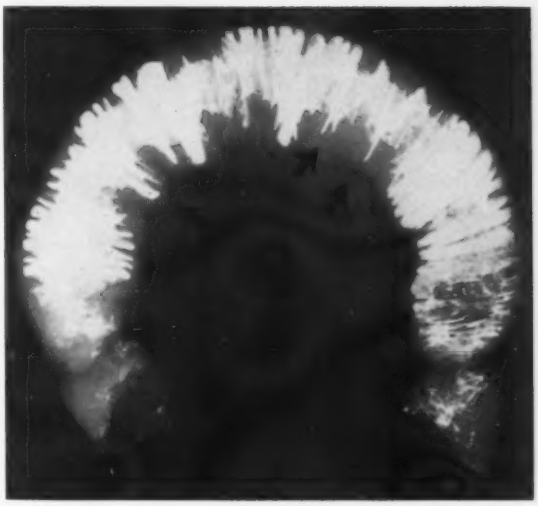

Fig. 7. Case 11. Roentgenogram of the resected segment of the small intestine after intraluminal distention with barium. The mucosal thickening, the narrowing of the lumen, and several spike-like projections (arrows) are well demonstrated.

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DUODENAL HEMATOMA INDUCED BY COUMARIN*

By JEROME F. WIOT, M.D., AARON S. WEINSTEIN, M.D.,† *and* BENJAMIN FELSON, M.D.

INTRAMURAL hematoma of the duodenum is an important and not uncommon entity which can be readily diagnosed by roentgenologic methods. Although described pathologically as early as 1894,[21] a detailed report of its roentgen aspects did not appear in the American literature until 1954, when Felson and Levin,[6] of this institution, described pathognomonic roentgen signs in 4 cases. Of the 23 proved cases found in the literature, 17 were the result of trauma.[2,3,6,7,11,15–17,21,22,25–29] In addition, 3 cases of spontaneous origin[3,18,19] and individual cases related to carcinoma of the pancreas,[19] pancreatitis,[15] and an obscure bleeding diathesis in an infant[9] have been recorded. Additional cases, similar pathologically, have also been noted as occurring elsewhere in the small bowel and in the colon.[12–14,18] Although intramural hematoma secondary to anticoagulant therapy has been described,[1,8,20] none of the reports mention roentgen contrast studies and none of the cases involved the duodenum.

Recently, we have encountered 2 patients with coumarin intoxication in whom roentgen changes in the duodenum resembling those previously observed in proved cases of intramural hematoma were seen. Since operation was not indicated in these cases, pathologic proof is lacking. However, in the light of the clinical and laboratory evidence and the subsequent spontaneous regression of the roentgen signs, the diagnosis can hardly be doubted.

REPORT OF CASES

CASE 1. E.L., a forty-one year old Negro male, was first admitted to the Cincinnati General Hospital on June 12, 1958 complaining of severe chest pain of three hours' duration. Physical examination was unremarkable except for a mild hypertension of 140/90. Laboratory studies, including serum amylase and a liver profile, were normal. An electrocardiogram revealed an acute posterior myocardial infarction. The patient was placed on a coronary occlusion regimen which included anticoagulants. The prothrombin time was maintained at about 20 per cent during hospitalization and on the twenty-eighth hospital day he was discharged on 5 mg. of coumarin per day.

Ten days after discharge he returned to the hospital complaining of cramping periumbilical pain, nausea, and vomiting of clear liquid for one week. On the day of admission the vomitus had become dark brown and the urine was grossly bloody. There was no history of trauma.

Physical examination on the second admission revealed a blood pressure of 120/100 and a pulse rate of 100. The temperature and respiratory rate were normal. He was in no acute distress. Positive findings were limited to the abdomen, which revealed generalized tenderness, most marked about the umbilicus.

The hematocrit was 33 per cent and the hemoglobin 10.6 gm. The white blood cell count was 11,000. The prothrombin time could not be determined, as a clot did not form. The bleeding time was seventy-five seconds and the clotting time was twenty-five minutes by the three tube method. Stool and vomitus were guaiac positive and the urine contained gross blood.

He was placed on an antacid, tincture of belladonna, and intravenous vitamin K. In approximately eight hours he stopped vomiting, and after thirty-six hours he was symptom-free. The prothrombin time gradually returned to normal during this period.

On the third day of the second hospital admission, an upper gastrointestinal series revealed marked changes in the mucosa of the entire duodenum distal to the bulb (Fig. 1, *A* and *B*). The mucosal folds appeared rigid and thick with the barium-filled troughs between them considerably narrowed. The normal feathery appearance was lost and the duodenal margins showed "spikes" of varying height. Longitudinal folds were not visible, so the mucosal

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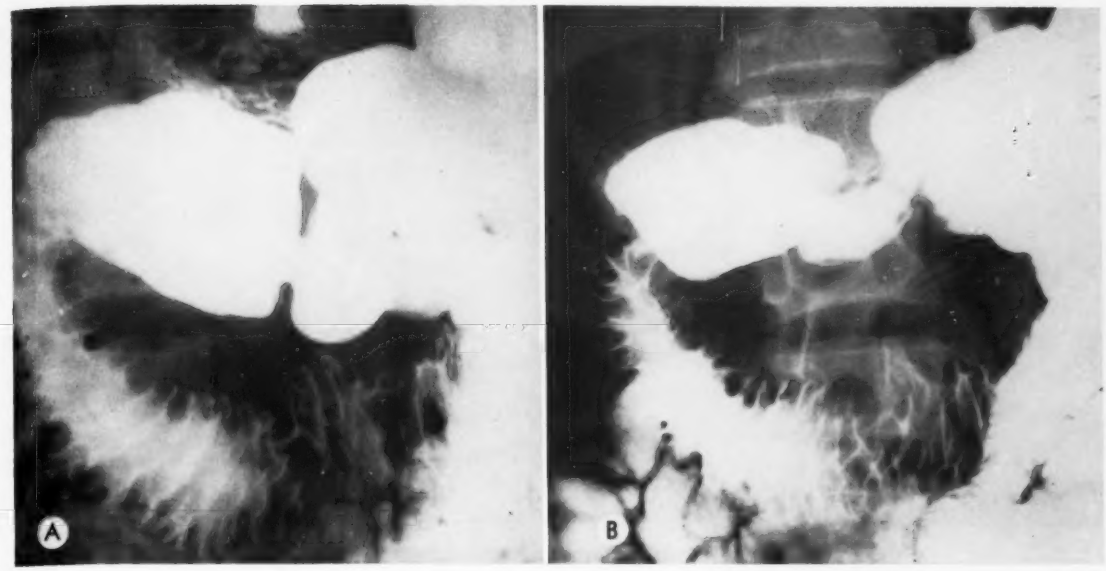

Fig. 1. Case 1. July 24, 1958. (*A* and *B*) There are marked mucosal changes of the duodenum, with rigid thickened folds and narrow troughs between them. The pattern simulates a "picket-fence." The extrinsic pressure on the duodenum was more apparent on other roentgenograms.

pattern had a "picket-fence" appearance. There was a suggestion of localized extrinsic pressure on the medial wall of the descending limb of the duodenum. There was no evidence of obstruction. A follow-up study one week later showed considerable improvement in the duodenal pattern (Fig. 2) and after another week the gastrointestinal series was entirely normal (Fig. 3).

He was discharged on the nineteenth hospital day on 5 mg. coumarin per day and remained well except for one bout of epigastric pain, which subsided when he discontinued the medication for four days. He was last seen at the Cincinnati General Hospital in June, 1959, at which time he was asymptomatic.

Case 11. (Reported through the courtesy of Dr. James H. Scatliff, Yale University School of Medicine.) W.H., a sixty-seven year old white male, entered the Grace-New Haven

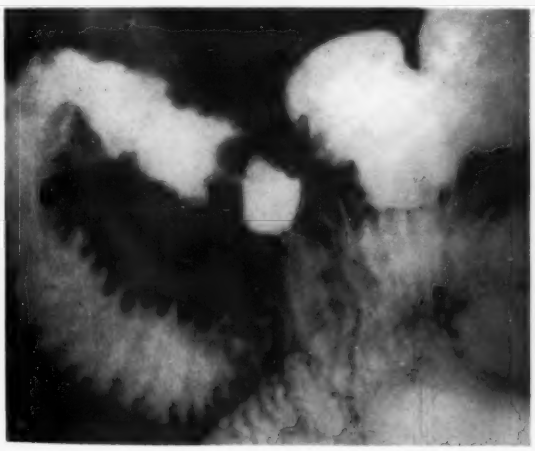

Fig. 2. Case 1. July 31, 1958. Definite improvement has occurred, but the mucosal pattern is still recognizably abnormal.

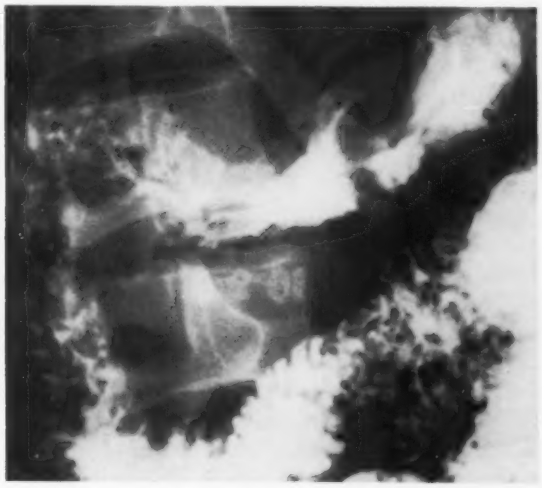

Fig. 3. Case 1. August 8, 1958. The mucosal pattern is now entirely normal.

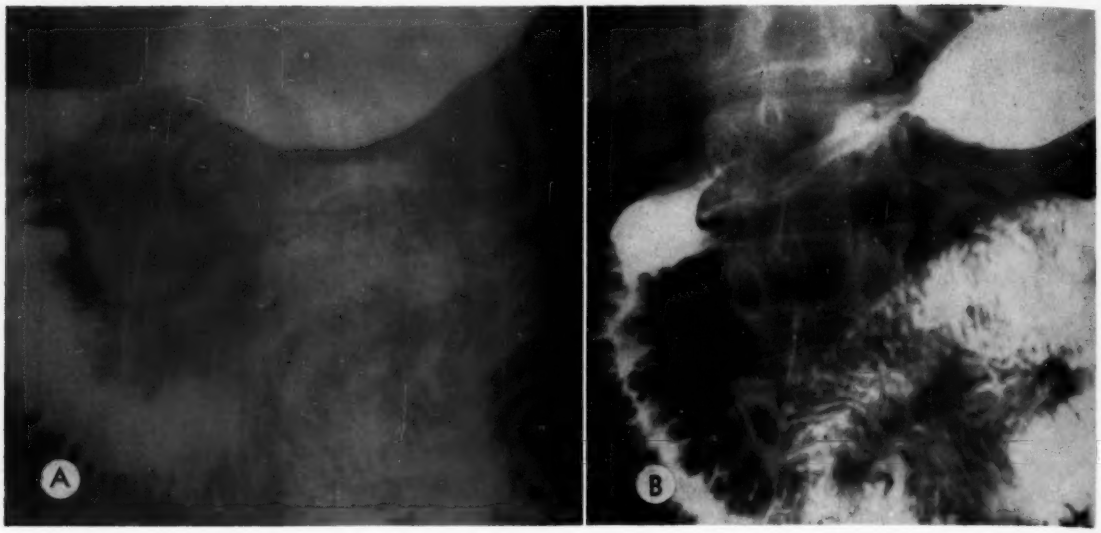

FIG. 4. Case II. December 1, 1959. (*A* and *B*) Abnormal mucosal pattern with "picket-fence" appearance, similar to the findings in Case I. Extrinsic pressure upon the greater curvature of the distal end of the stomach is evident.

Community Hospital, November 24, 1959, with a history of bleeding into the skin and eye, pink urine, and black stools for about ten days. Lower abdominal pain had been present for several days. He had had an acute myocardial infarction in February, 1959, since which time he had been maintained on anticoagulants. Shortly before onset of the bleeding he had received overdoses of coumarin by mistake.

Physical examination revealed ecchymoses in the skin, conjunctival hemorrhage, and petechiae in the palate. His abdomen was slightly distended and tense, with mild tenderness of the right costovertebral angle.

The prothrombin time was greatly increased. The hematocrit was 29 per cent. Liver profile studies and serum amylase were normal. The electrocardiogram showed evidence of an old anterolateral myocardial infarction. A stool sample was reddish brown in color and guaiac positive. Anticoagulant therapy was discontinued and he was given two units of packed red blood cells, which resulted in prompt recovery.

An upper gastrointestinal series eight days after admission showed a striking swelling of the mucosal folds of the duodenum (Fig. 4, *A* and *B*). The troughs were narrowed and spiked. As in Case I, longitudinal folds were lacking, resulting in a "picket-fence" pattern. The involved area was moderately distensible, and there was no obstruction. There was evidence of mild extrinsic pressure on the medial aspect of the descending duodenum and on the greater curvature of the distal end of the stomach. A barium enema examination was normal.

An upper gastrointestinal series thirty-eight days later showed a completely normal duodenal pattern with no evidence of extrinsic pressure on the duodenum or stomach (Fig. 5).

DISCUSSION

The roentgenographic changes of intramural hematoma of the duodenum are felt to be diagnostic when all of the manifestations are present. Classically, the lesion involves the descending and transverse segments of the duodenum but occasional cases have also involved the bulb and/or upper jejunum. The proximal portion of the abnormal segment shows only thickening of the mucosa. More distally, an intramural mass is present, associated with widening of the lumen and a coil-spring appearance due to crowding of the valvulae conniventes. Partial obstruction is common. Frequently, extrinsic pressure upon the greater curvature of the stomach and widening of the duodenal loop are seen, probably attributable to extraduodenal extension of the hematoma.

The findings in both of our cases differed somewhat from this description in that

there was no evidence of a localized intramural mass or obstruction, and a distinct coil-spring appearance was not apparent. Mucosal swelling and extrinsic pressure upon the stomach and duodenum were the only abnormalities. It is possible that the absence of an intramural mass is the result of failure of clot formation related to the coumarin therapy, or perhaps the diffuseness of the hematoma. The rapid clinical and roentgenologic recovery following medical treatment in our cases is consistent with the course of previously reported cases of unoperated intramural hematoma.

Felson and Levin[6] attempted to reproduce the roentgen findings of intramural hematoma of the duodenum experimentally by injecting water into the wall of a segment of normal small intestine removed at autopsy. They were able to duplicate the mucosal changes and intramural mass seen in clinical cases of intramural hematoma but not the coil-spring appearance (Fig. 6). The mucosal changes which they demonstrated experimentally very closely resemble the findings encountered in the present cases.

The association of hemorrhage with anticoagulant therapy is relatively frequent. In response to a questionnaire submitted to 228 physicians by Russek and Zohman[21] in 1953, 45.6 per cent reported one or more instances of serious hemorrhagic complications with 122 deaths from anticoagulant therapy for a variety of conditions. Thirty deaths from gastrointestinal hemorrhage were recorded, and this complication was second only to cerebral hemorrhage in frequency. Two-thirds of the deaths from gastrointestinal bleeding were attributed to pre-existing peptic ulcer but, in the remaining 10, the source of hemorrhage was not found.

FIG. 5. Case 11. January 7, 1960. Normal duodenal pattern.

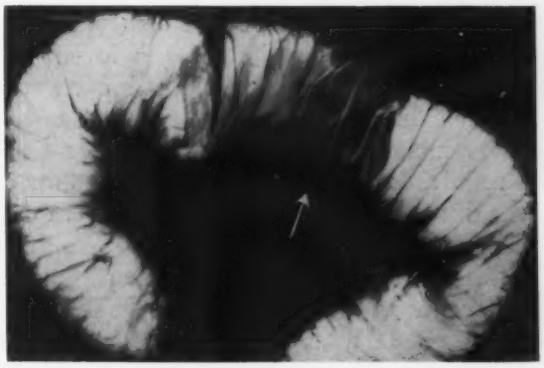

FIG. 6. Experimental attempt to duplicate the roentgen appearance of intramural hematoma by injection of water into the wall of a closed segment of jejunum filled with barium. The pattern resembles very closely the changes seen in Figures 1, *A* and *B*, and 4, *A* and *B*. (Reproduced with permission of *Radiology*.[6])

The mechanisms of hemorrhage in coumarin therapy are complicated. Briefly, these have been divided by Peyman[22] into four categories: (1) disturbance of normal blood clotting; (2) toxic effect on the capillary walls; (3) impairment of the normal adhesiveness of the platelets; and (4) accentuated recanalization of thrombosed arteries and veins. Not only is there great variation in the amount of the drug required to produce intoxication among different individuals but also in the same individual at different times.

The frequent occurrence of intramural hemorrhage in the duodenum as opposed to other areas of the bowel, in cases of trauma, is attributed to the fixation of this portion of the intestine at the ligament of Treitz with the trauma forcing the fixed retroperitoneal portion of the duodenum against the spine. The history of trauma,

however, must often be diligently sought, for in many reported cases this important fact was not uncovered until after the surgical findings.

As mentioned earlier, there have been 6 reported cases of intramural hematoma of the duodenum in which a history of trauma could not be obtained. In addition to the 2 cases secondary to coumarin, we have recently seen 3 other cases of duodenal hematoma in which trauma apparently did not play a role. The first of these was an elderly man with a very low prothrombin time secondary to alcoholic cirrhosis. The second was a nineteen year old boy with hemophilia. Both of these patients showed changes in the duodenum identical to those in the present report. The third patient, who also denied trauma, showed the classical roentgen findings described by Felson and Levin.[6] Thus, a total of 11 cases of duodenal hematoma in which a history of trauma could not be elicited have been documented. However, trauma as a factor in some of these cases cannot be excluded since, in the presence of a bleeding tendency, a minor unrecognized injury may have been sufficient to initiate the process. It is of interest that the last patient mentioned above and 1 of those reported by Felson and Levin were butchers. Possibly trauma to the abdomen occurred while they were carrying large slabs of meat, an everyday event in this occupation.

The reason that spontaneous hemorrhage so commonly occurs in the duodenal region of the gastrointestinal tract is obscure. However, there appears to be such a predilection, since a number of cases of diffuse duodenal hemorrhage have been reported in association with severe burns, tumor and trauma of the central nervous system, and major operative procedures. Recently, Katz[10] reported autopsy findings of "hemorrhagic duodenitis" in 25 cases of myocardial infarction. Although most of these patients were receiving anticoagulants immediately prior to death, none were apparently intoxicated by the drug. Roentgenograms were not available in any of the cases.

In the absence of roentgen evidence of an intramural mass or coil-spring appearance, the differential diagnosis of duodenal hematoma, as represented in the 2 cases in the present report, must include infarction secondary to vascular occlusion and such inflammatory conditions as pancreatitis and acute duodenitis. In these conditions the duodenal wall may be infiltrated by inflammatory exudate or hemorrhagic edema, resulting in a roentgen appearance similar to that caused by coumarin. The differential diagnosis is extremely important, as operation in the presence of an unrecognized bleeding tendency might have dire results. The history of coumarin administration, the low prothrombin time, the typical roentgen findings, and the response to therapy make the diagnosis of intramural hematoma caused by anticoagulants virtually certain.

SUMMARY

The roentgenologic appearance of intramural hemorrhage secondary to anticoagulant therapy has not been previously described despite the relatively frequent occurrence of gastrointestinal and other hemorrhagic complications of such therapy. The roentgen features differ somewhat from those of intramural hematoma caused by trauma, possibly because of its more diffuse nature and/or the absence of clot formation. Although the striking findings are also consistent with inflammatory changes in the duodenum, the history, laboratory, and roentgen findings readily establish the diagnosis.

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We are indebted to Dr. James H. Scatliff, Yale University School of Medicine, for permission to report Case 11.

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NOTES ON A SMALL BOWEL EXAMINATION*

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ELEVEN years ago Weintraub and Williams[8] described a method for a rapid small bowel examination using iced barium and saline solution. Since then, a similar method has been used by the author with very satisfactory results. Inquiry among my colleagues indicates that the technique is little used and is not well known. It is being described again because of its advantages, which are as follows: (1) areas of disease are easier to find; (2) the patient is examined in a single visit to the department, and in less time; and (3) by examining an area which frequently is bypassed, additional diagnoses are established.

TECHNIQUE

Barium of the suspended type (micropaque [R] is preferred), in the amount of 70 gm. in 200 ml. of ice cold tap water, is employed to examine the hypopharynx, esophagus, stomach and duodenum. The relative amounts are not important and can be varied to suit personal preference. On completion of the upper tract examination, note is made of the amount of barium remaining in the stomach, for in some patients the entire small bowel and colon will fill in thirty minutes. With practice it can be determined when these patients should be called back before the lapse of thirty minutes.

A glass of ice cold normal saline solution is then given every fifteen minutes until the examination is completed. The saline is made from tap water and table salt in the department (about 200 ml. per glass). It must be cold and should be isotonic for fastest transit time. Nauseated patients are placed on their right side on a hospital cart. Nausea is additionally controlled by inhalation of aromatic spirits of ammonia U.S.P. on a cotton pledget. It is well to explain to the patient that drinking the saline solution will shorten the examination and aid in elimination of the barium later. Experience with constipation after being examined with the older nonsuspended barium makes patients react more cooperatively to this procedure. We do not find it necessary to give the ambulatory nonconstipated patient a laxative with this technique.

Thirty minutes before completion of the stomach examination, the average patient is briefly fluoroscoped and a roentgenogram is made. Abnormalities requiring additional study are noted. Stasis, retroplacement of loops behind the spine and other changes are identified and disposition is made of the patient. If the barium has reached the cecum the examination is terminated; 31 per cent of the patients fall into this group. In an additional 34 per cent of patients, the barium is found in the terminal ileum. Reexamination of the latter group is made at fifteen to thirty minutes to study the ileocecal area. In 18 per cent the barium will have reached the midileum at thirty minutes and usually will have passed into the colon.

When passage of the barium is slower than this, the patient is evaluated at thirty minutes for the cause of delay.

Patients on a salt free diet because of nephritis, heart failure or other reason, should be given plain ice cold water. It is not as efficacious as the saline solution, but considerable motility still can be induced. Patients who have salt retention may gain 2–5 pounds if given the saline solution.

Occasionally more barium is required to study some questionable area, to relate various areas of compartmentation, or to confirm a serious lesion.

Patients are requested not to empty the bladder during the first thirty minutes or

* Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.

until after they have been fluoroscoped. It is much easier to study the pelvic loops with the bladder filling the pelvis and elevating the small intestine.

ADVANTAGES

1. Areas of disease are easier to find. Stress demonstrates variations of pattern and motility and elicits pain in the abnormal small intestine. Polyps intussuscept with a slow forward motion and a quick plunger-like return which is very characteristic when once identified. Unsuspected cancers are revealed when barium backs up behind them (Fig. 1). Parasites such as *Ascaris lumbricoides* first show as negative defects and then usually ingest barium at forty-five minutes. Foreign bodies and enteroliths can be located for more detailed study. Peritoneal adhesions are indicated by increased deformation with rapid peristalsis.

Fig. 1. Primary carcinoma of the jejunum in a seventy year old woman with blood loss anemia. Note stasis behind the lesion (arrow). Deep gastric folds were confusing initially.

Fig. 2. Pancreatitis with mass (arrow), edema of loops and displacement of jejunum.

2. The method is convenient for the patient and the radiologist. Diagnoses are established and reported in a shorter time.

3. Unsuspected disease is discovered. Space occupying lesions are easier to evaluate when the small intestine is filled with barium (Fig. 2 and 3). Tumors of solid organs, aneurysms, and intraperitoneal and extraperitoneal lesions are outlined by the opacified intestine. We routinely combine excretory urography with a rapid small intestinal series for evaluation of paraaortic and iliac lymph node areas in lymphoma patients.

DISADVANTAGES

1. Ulcers are harder to see. When first using suspended barium, the radiologist is troubled by the necessity for more meticulous study of the mucosal pattern, as the craters are harder to identify. In our opinion, this is of little importance, for good

FIG. 3. Gas filled pocket (arrow), due to abscess following diverticulitis, visualized by filling small bowel loops.

spot roentgenography is always necessary in order to find many of the ulcers.

2. Narcotics will delay motility. If patients are seen within twenty-four hours after the administration of morphine, demerol or similar narcotics, they have protracted transit times. A decision must be made as to the probable value to the patient of the small intestinal study and, if the indications are not urgent, the examination should be terminated and resumed on another day.

FINDINGS AND COMMENTS

HERNIAS AND CONGENITAL CHANGES

The viscus occupying a hernial sac can be established and internal hernias recognized by this examination (Fig. 4). It is not rare to find femoral, inguinal or umbilical hernias which good clinical examiners have missed. Twice in infants we have observed umbilical hernias too small to be clinically identified. In the recognition of internal hernias, attention should be paid to agglutination of loops, recognized by the palpating hand, and to compartmentation (Fig. 5).

The retroplacement of loops behind the spine, seen first at the thirty minute recumbent study and confirmed by upright lateral roentgenograms, has been valuable in our experience.[5] This sign is present with paraduodenal hernias and adhesive bands with considerable regularity (Fig. 6). Admittedly, the number of such findings is not large, but the patients suffering from this compartmentation are likely to suffer for years unless there is a careful small bowel study.

Vestigial mesenteric attachments are particularly difficult to recognize without good roentgenograms. Only a slight straight-

FIG. 4. Congenital failure of rotation with the jejunum displaced to the right and without a normal relationship to the superior mesenteric artery. The patient had been admitted twice in five years for recurrent obstruction.

ening or deflection of the loops may be observed. If the patient is emotionally stable and if the complaints are related to meals, it is worthwhile to repeat the examination when there is the merest suspicion of compartmentation with abnormal position of loops.

Multiple diverticula are of some clinical significance in macrocytic anemia. Reduplications and cysts can be demonstrated in relationship to bowel segments if all the loops are filled on one or two roentgenograms (Fig. 7).

INTRINSIC VASCULAR DISEASE

Although our clinical colleagues may require some persuasion, we find that ingestion of barium is no more dangerous than of food for cases of the acute mesenteric vascular accident. Recognized clinically by the acute onset, local mass and bleeding, the extent and improvement of the mesenteric occlusion can be demonstrated roentgenographically (Fig. 8 and 9). Judgement must be exercised in determining the best time for small bowel study, but knowledge of the behavior and size of the lesion improves its management. All cases do not require surgical intervention.

Obstructions of significance can be diagnosed from barium enema studies alone, and without oral barium. We have occasionally used hyperhydration of the patient with saline solution to prevent obstruction of the colon when, by accident or design, oral barium has been given.

Fig. 6. Lateral upright view of the same case as Figure 4 showing loops (between arrows) behind the spine.

Fig. 5. Compartmentation by omental adhesion in the deep pelvis, presumed due to appendectomy in childhood. Recurrent vomiting, pain and bleeding forced surgical exploration after intermittent attacks over a three year period. The condition had been identified three years before surgery but the patient and surgeon initially were unwilling to accept the diagnosis.

FIG. 7. Mesenteric cyst causing displacement and compression of the bowel and with the midabdominal loop of the small intestine (between arrows) too intimately bound to the mass to be preserved surgically. This is one of 3 such cases identified in the past six years, each of which had one midabdominal loop of the small intestine that required removal.

DISORDERED MOTILITY

Reference should be made to the work of Golden[3] who coined this useful term. Insofar as we can determine, in our examinations the same patterns are identifiable as those described by him, allowing for a condensation of the time internal. It seems more logical to stimulate the intestine to maximum effort than to fast the patient. Functional bowel states related to social maladjustment can be demonstrated to have irregular patterns of stasis, edema and hypermotile segments identical to those described by Friedman.[2] Depending on the interpretation, similar changes may be attributed to allergy, edema, nervous states or nutritional causes (Fig. 10). Generally, in malabsorption syndromes of the sprue type the barium moves slowly, so these cases can easily be distinguished from the other group. Some care should be taken to exclude electrolyte imbalance, as in heart, liver or kidney disease and anemia, as the cause for the delay. Insofar as we can determine, this differentiation is clinical and requires laboratory confirmation but it may be initially suggested by the gastrointestinal study.

PEPTIC ULCERS

A very interesting relationship exists between peptic ulceration, particularly with duodenal ulcers, and motility. Some patients show very rapid and others very slow transit times. At thirty minutes the barium had reached the rectum in almost twice as many ulcer patients as patients without demonstrable craters. No rate difference was found between those having only deformity of the cap, without demon-

FIG. 8. Acute vascular occlusion (arrow) in the terminal ileum. This was treated conservatively with complete return of the pattern to normal after a clinical diagnosis.

strable ulcers, and those without any evidence of ulcers in this respect. Of 514 ulcer patients having demonstrable craters, the barium had reached the rectum in 3.1 per cent, whereas, of the remaining 1,236 patients, it had reached the rectum in only 1.8 per cent. This is not a new observation, according to Alvarez.[1] The slow transit times are complicated problems related to perforation, inflammation and duodenal ileus. In 10.7 per cent of the ulcer group, barium did not pass the jejunum at thirty minutes as compared to 2.3 per cent of the non-ulcer group.

An impression is gained from the pattern of repeat examinations that patients with average transit times are easier to control and do not return for repeat examination when released from medical management. Sometimes a patient with an initially slow transit time becomes hypermotile as the ulcer heals. Others revert from hypermotility to average transit times with healing of the ulcer. The patient without evidence of hypermotility appears unlikely to have a serious duodenal ulcer.

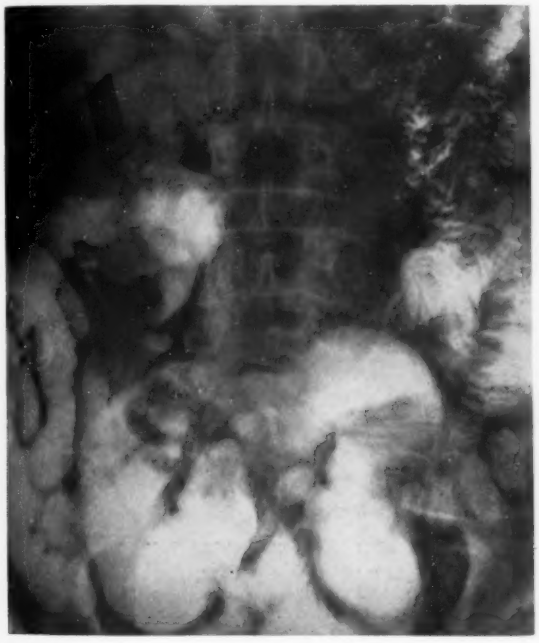


Fig. 9. Acute vascular occlusion (arrow) of undetermined cause. Exploratory laparotomy six months after this study showed residual adhesion between two loops; resection was not necessary.

Fig. 10. Illustration of the rapid transit of barium to the sigmoid colon (fifteen minutes) in an adolescent girl; the cause was not recognizable clinically.

Attention was strongly called to this hypermotility relationship by fluoroscopy of the first patient with a recognized Zollinger-Ellison syndrome. She had had diarrhea for many years with copious stools several times a day before the acute ulcer pain developed. A similar patient has been described with watery diarrhea and islet cell tumor without peptic ulcer.[7] Parenthetically, note should be made of the difficulty in adequately studying the jejunum in such cases. The continuous voluminous gastric hypersecretion dilutes the barium rapidly, making a satisfactory mucosal pattern study very difficult, if not impossible. The patulous, slightly obstructed atonic cap and second portion of the duodenum add to the problem. Unless extreme care is taken, the jejunal ulcer will be hidden by the stomach and adjacent jejunal loops. Changes in caliber of the lumen and

FIG. 11. This patient had a cast syndrome with a high degree of small intestinal obstruction at the duodenojejunal junction (arrows). It was postulated that marked edema was present from the appearance of the few jejunal loops seen at forty-five minutes. Edema was thought to be the cause of obstruction and a positive indication for surgery, which confirmed the roentgen findings. Rapid and complete recovery occurred.

sharp kinks or knuckles of small intestine should be especially watched when typical gastrosuccorrhea and duodenal ileus are encountered.

We have speculated that the rapid gastric emptying rate and the hypermotile small intestine are reverse facets of the hypersecretion factor in ulcer genesis; *i.e.*, peptic and acid secretions are induced by the transit of food through the small intestine without sufficient food remaining in the stomach to protect the lining and to neutralize the enzymes and acid formed. In other words, peptic ulcers occur either in patients with hypersecretion or in those who empty their stomach too soon to neutralize the normal secretions. If this be true, routine measurement of motility would provide useful clinical information with indications for either anticholinergic drugs or antacids in the management of ulcer patients.

Other conditions associated with hypermotility in our material are occasional cases of hyperthyroidism, emotional instability, gallstones and nonspecific acute enteritis.

INFECTION

The constitutional reaction to infection has many interesting relationships. Some patients with duodenal ileus from the superior mesenteric artery syndrome appear to have had recent changes in nutrition or in protein or electrolyte balance related to infections immediately preceding the development of acute symptoms. A seventeen year old boy, in a body cast, was rapidly deteriorating without much fever but with leukocytosis, vomiting and electrolyte imbalance. His original injury was a fracture of the femur four months before, treated by an intramedullary nail. Osteo-

FIG. 12. Persistent distention and compression of the terminal ileum (arrows) due to Jackson's membrane found at surgery.

myelitis developed and, on admission here, the pin was removed and a body cast applied. A duodenal ileus followed. The interesting feature is, however, that the distal edematous loops were recognized on the roentgenogram and verified at surgery (Fig. 11). After duodenojejunostomy, the postoperative course was smooth.

Less dramatic episodes are sometimes seen with the gradual development of duodenal ileus after an infection or diarrhea which has been treated with antibiotics. We have found such a patient to have normal duodenal motility prior to such an infection, only to discover, a few months later, a typical "superior mesenteric artery syndrome." At times the unfortunate patient with chronic peritonitis and adhesions seems to undergo similar obstructive episodes after infections. An old established regional ileitis similarly may be adversely affected by respiratory infections. Such episodes of recurrent inflammation have been noted by Parsons,[5] who attributes them to edema from adhesions, with eventual obstruction.

Fig. 13. Distended small bowel loop of the ileum (arrows) in a patient who had had colicky complaints throughout her life. Patient was of small stature and slight habitus. Operation was refused. This loop was seen to be rotated medially and laterally and to be inverted in the midabdomen on each of four examinations over a five year period.

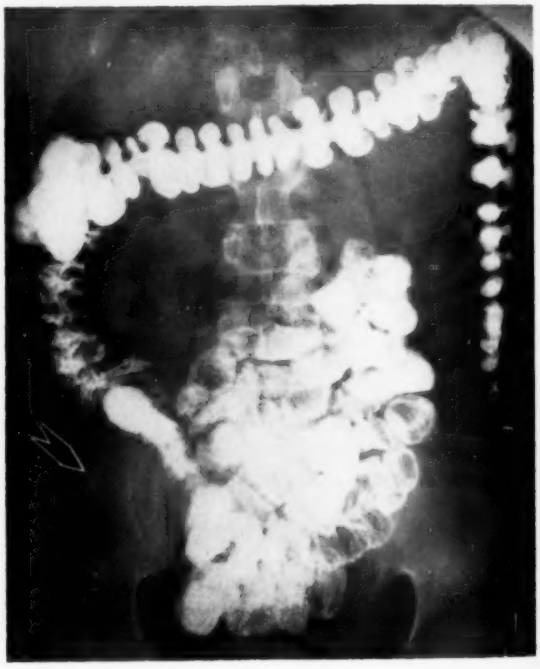
Fig. 14. Subacute small bowel obstruction (arrows) from intestinal adhesions and pelvic peritonitis. Since the patient tolerated food, barium was given to identify the obstruction and the amount of disturbance of the intestinal tract. A single band at the terminal ileum was found at surgery.

CECAL AREA LESIONS

We find with Golden,[3,4] that the cecal area can profitably be studied from above for such diverse conditions as Jackson's membrane (Fig. 12), congenital bands and webs, short patches of obstruction from many causes (Fig. 13 and 14), ileitis (Fig. 15 and 16), appendicitis (Fig. 17), abscess, granulomas and tumors. Extrinsic tumors of the appendix and cecum and enlarged iliac lymph nodes can protrude into the barium column to give graphic evidence of their existence (Fig. 18). One case of intussusception of the ileal cecal valve through a tight submucosal constriction was found and later relieved by surgery. Another case of ileal intussusception in an eleven year

FIG. 15. Early thickening and edema of the terminal ileum (arrow), thought to be due to regional ileitis in the developing stage. This is a not uncommon finding that requires a small bowel examination for verification as it is a diagnosis of exclusion, since only this minor change is present.

old boy with healed duodenal ulceration was almost accidentally found at the small bowel study when there had been no prior clinical or roentgenologic suspicion. No polyp or diverticulum to explain the intussusception was found at surgery and he has been well since then. We suppose this to



top right

FIG. 16. Advanced stage of regional ileitis (between arrows) with cecal involvement.

bottom right

FIG. 17. An organized appendiceal mass of five years' duration (arrow) is outlined by small bowel loops. The diagnosis, not established by previous examinations made elsewhere of the gastrointestinal tract, was verified surgically.

Fig. 18. Carcinoma of the colon with extraluminal mass (arrows) slightly obstructing the small bowel. The obstruction had not been clinically identified. The barium enema study was not diagnostic but this appearance of small bowel obstruction and the demonstration of the mass between the cecum and ileum by compression studies etablished the need for surgery.

be another example of hypermotility with duodenal ulcer, for we are certain that an ulcer was present a year earlier.

None of these cases are common enough to lead to directed examinations by the clinician but taken together they make up a respectable number of serious diseases discovered by routine examination.

SUMMARY

A technique for the physiologic forcing of barium through the small intestine at a rapid rate is again presented in the hope that, because of its simplicity and usefulness, it will be more widely adopted. The entire small bowel can be visualized in one hour in the normal patient with, at most, two roentgenograms. The discovery of unexpected disease and guides for therapy are enough to justify the routine use of the method. The adoption of adequate gonadal protection, rapid screening, fast screens and film, high kilovoltage and modern developers more than compensates for the possible additional exposure and makes radiation dosage tolerable. The time saved should alone justify the additional expense of suspended barium, without considering the scientific reasons for its use.

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MUCORMYCOSIS OF THE COLON

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MEMBERS of the *Mucoraceae* family of fungi are common saprophytes and laboratory contaminants. They have been recognized as pathogens in man, on occasion, since the last century.[12] Prior to 1954, accounts have been rather rare; since that time, however, articles of infection in man have been numerous. Most of these have been concerned with involvement of the paranasal sinuses, central nervous system, or the lungs. Less common have been reports of cases involving the intestinal tract. Within the past three years, 4 cases have been discovered at this institution which will be reported elsewhere. One of these, in which the colon is involved, is reported below.

REPORT OF A CASE

A fifty-one year old white male was admitted to Ellis Hospital in October, 1956, three months following circumcision for an ulceration of the prepuce of the penis. At admission he complained of early fatigue and slight epigastric "burning" sensation. On physical examination there were present bilaterally-enlarged inguinal lymph nodes, small axillary lymph nodes, a healed furuncle in the right axilla, and moderate spleen enlargement.

Initial blood studies revealed: red blood cells 2.4 million; hemoglobin 58 per cent, 9 gm.; packed cell volume 28 per cent; platelets 78,000; white blood cells 2,000 with stabs 2 per cent, segm. 16 per cent and lymphocytes 82 per cent; slight anisocytosis; and immature and atypical lymphocytes. The bone marrow study demonstrated chiefly primitive cells having the characteristics of lymphoblasts and an increase in mature lymphocytes. Erythroid tissue was diminished but adequate. Rare megakaryocytes were seen. On review of the prepuce section, the corium was noted to be infiltrated by lymphoid and plasma cells, few eosinophils, and many large mononuclear cells in forms of distinct nodules or gathered about blood vessels and nerves. The impression of both was lymphoblastic lymphoma.

The patient was given one transfusion and treated with ACTH, 20 units by intravenous drip daily for ten days, and then 30 mg. of meticorten. Within a few days his white blood cell count dropped and remained between 400–800 cells. Radiation therapy was given to the splenic area with a surface absorbed dose of 1,427 rads. Roentgenograms of the spine, chest and intestinal tract were negative except for a few small diverticula in the descending colon (Fig. 1).

The patient was discharged after three weeks and maintained on meticorten for seven months. During this period he received several units of packed cells, testosterone and "spray" radiation of 105 r exposure dosage on two occasions.

He was re-admitted in June, 1957 with complaints of increasing fatigue, weakness, chills, fever, abdominal cramps, left lower abdominal pain, and pain at the sites of upper incisor

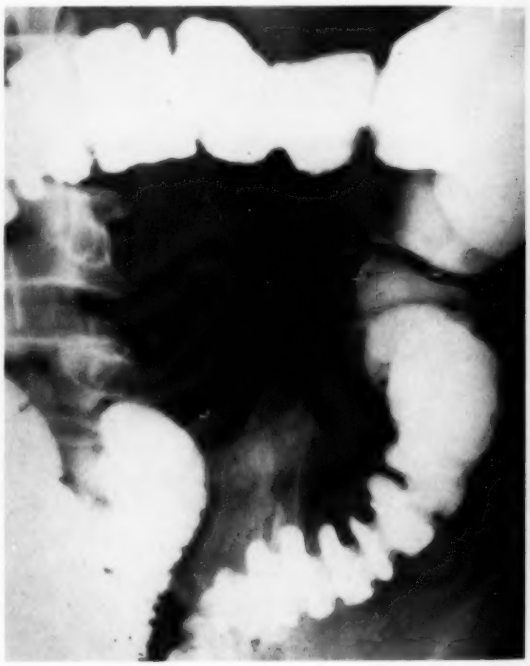

FIG. 1. Barium enema examination in October, 1956 was negative except for several small diverticula of the descending colon just above the iliac crest level.

periapical infections and cellulitis. He was tender in the abdominal left lower quadrant but no spasm or rigidity was noted.

The teeth were extracted and the abscesses were drained without complication. Abscess culture and blood cultures were sterile. Large amounts of penicillin, streptomycin and chloromycetin were administered. A barium enema study (Fig. 2 and 3) revealed an annular defect of the descending colon outline where diverticula were noted previously. There was a short zone of involvement and abrupt transition to a normal outline. A few diverticula were seen in the area. The bowel was not fixed in position during fluoroscopy. Because of the history, lymphomatosis involvement was considered and an estimated dose of 1,500 rads was delivered to this zone. He was discharged in ten days at which time there was no temperature elevation.

Final admission followed in one week because of no bowel movements and increasing severity of abdominal pain which was aggravated by movement. He had chills and a spiking fever up to 105° F. The abdomen was distended, tympanitic, and there was a palpable mass in the lower left quadrant. At this time, his red blood cell count was 2 million, hemoglobin 6.7 gm., platelets 70,000 and the white blood cell count 150. A roentgenogram of the abdomen showed intestinal obstruction at the descending colon. Penicillin, chloromycetin, streptomycin and hydrocortisone therapy was continued.

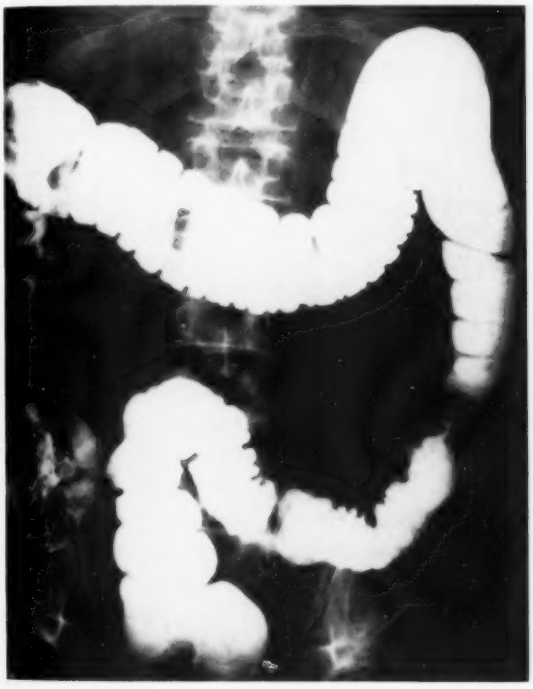

Fig. 2. Barium enema examination in June, 1957, about two weeks prior to laparotomy, revealed a short, irregular, annular narrowing of the colon at the same level. Two diverticula were noted on the lateral aspect of the colon at the margin of the lesion.

Fig. 3. Enlargement of involved portion of Figure 2.

Although he was considered a poor surgical risk, after three days a laparotomy was performed. Foul-smelling material was encountered and portions of necrotic descending colon wall were recognized. The area was drained and the abdomen closed partially. The patient became very weak and expired on the third day.

At postmortem examination 300 cc. of clear amber fluid was removed from the peritoneal cavity. There was thickening and marked inflammation of one short area of the descending colon. In dissecting the colon in this zone, a mass of fecal material was found in the mesocolon. The peritoneum at the site of surgery and around the colon was inflamed and showed yellowish fibrin pus deposits. On microscopic section (Fig. 4) there were necrosis and leukocytic infiltration, extensive submucosal hemorrhage and thrombosed vessels. The thrombi were formed by a meshwork of hyphae and red blood cells. Hyphae, in masses, were growing through the vessel walls into adjacent tissue which was infarcted. These had the typically wide diameter of mucormycosis, were

Fig. 4. Photomicrograph of section showing hyphae growing into the colon wall.

nonseptate and coenocytic, with branching almost at right angles. No cultures were made.

A secondary branch of the pulmonary artery was occluded by a thrombus formed by masses of the same type of fungus. These had grown into adjacent lung tissue where nearby were foci of bronchopneumonia.

There were a few superficial denuded areas in the mucosa of the stomach, but no fungi were found at those sites. The spleen had two small infarcted areas. The sinusoids were wide and focal interstitial hemorrhage was present. Normal bone marrow cells were replaced almost completely by atypical immature large cells with large vacuolated nuclei containing chromatin resembling lymphoblastic cells. Only a few red blood cells were present. The polychromatophilic cells of the granulocytic series were absent almost completely. The diagnosis was lymphoblastic leukemia.

No fungus invasion of any organs other than the colon and lung was found.

DISCUSSION

Mucoraceae hyphae are wide, with diameters ranging from 4–20 μ, nonseptate and having numerous branches, often extending outward at right angles.[10] Genera cannot be identified except by culture and usually this is not done prior to tissue fixation, since the presence of the fungus is not often suspected. Where cultures have been made, a member of the genus *Rhizopus* was usually found to be the responsible organism.[8,11]

Spores of the organisms are air borne and are believed to enter the body by either inhalation or by way of the intestinal tract, although spores are not found in the body. There is a strong tendency of the fungi to invade blood vessels and produce intense, localized inflammatory reaction, necrosis and thrombosis. The favored route of spread is along blood vessels or lymphatics, especially, in the perineural zones. In some instances only one system is involved, but, in almost as many, the disease is disseminated.

In a great majority there is an associated chronic, debilitating disease, usually leukemia or diabetes, and also, less commonly, nephritis, tuberculosis, liver abscess and diarrhea.[8,16] Several case histories indicate no such association.[5,14] The disease has been produced experimentally in animals in the acute toxic phase of alloxan diabetes.[4,9] The sudden increase in the number of reported cases has coincided with the

use of large amounts of antibiotics and cortisone, and the latter, especially, appears to be a significant factor affecting both defense mechanism and fungus spread and proliferation.[3,9]

Because of the production of thrombi in significant vessels by hyphae masses, the survival time of those patients treated to prevent bacterial infection may be less than of those succumbing to bacteria. The reports of deaths indicate only a very short period of survival from the time of onset of the disease.

Five cases with involvement of the intestine have been collected by Baker *et al.*[2] in addition to reported single cases.[6,7,13,17] The palate or esophagus was involved once. The stomach was involved in 2 patients, the small intestine in 3 and the colon in 4. In 1 case the disease was disseminated. The appearance of the lesion of the cecum in Clark's[6] diabetic-leukemic patient was described, following a gastrointestinal series, as consistent with a carcinoma or leukemic infiltration of adjacent lymph nodes.

When pathologic findings are discussed, the presence of intense inflammatory reaction with bowel wall edema, exudate and entrapped leukocytes is described. There is a sharp demarcation between involved and normal intestine.[15] Necrosis or ulcerations always are present with thrombosis in adjacent vessels. Hyphae are found growing from bowel wall into submucosal vessels and in the thrombi.

The sequence of events in this reported case cannot be established, however, with the short zone of involvement; with the presence of adjacent uninvolved diverticula, it may be assumed that perforation due to thrombosis or hyphae penetration was the initial event rather than perforation of a diverticulum.

This case emphasizes the error of irradiating a lesion without biopsy after large amounts of cortisone have been given, when there is a marked depression of the white blood cell count and a diagnosis seemingly has been established.

In similar cases with bowel defects and suspicion of perforation, prompt surgery may be indicated rather than permitting more infarction and spillage during a valueless delay. With the increasing use of massive therapy in diabetic or leukemic patients, more such cases are predicted.

SUMMARY

1. A case is reported of a patient with the diagnosis of lymphatic leukemia receiving large doses of ACTH and antibiotics who developed an annular lesion of necrosis and perforation of the descending colon due to mucormycosis.

2. Since the use of such therapy is increasing, a marked rise in the incidence of mucormycosis is predicted in patients with diabetes, leukemia and in those chronically ill, possibly with similiar types of intestinal defects.

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THE ROENTGENOLOGIC FEATURES OF CARCINOMA IN CHRONIC ULCERATIVE COLITIS*

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THE occurrence of carcinoma in chronic ulcerative colitis is a well-established fact; numerous authors have called attention to this relationship.[1–3] Less well known are the roentgenologic features of the carcinoma associated with ulcerative colitis. In order to clarify the roentgenologic manifestations of this disease and to explore the factors that may permit roentgenologic differentiation between carcinoma and stricture or polypoid hyperplasia in chronic ulcerative colitis, 49 cases of roentgenologically suspected carcinoma associated with ulcerative colitis were reviewed. Of the 49 patients, 27 were men and 22 were women.

A total of 29 of the original 49 patients were ultimately shown to have carcinoma of the colon superimposed on chronic ulcerative colitis. In all 29 cases the diagnosis was proved by pathologic examination. Among the other 20 cases in which there was roentgenologic suspicion of a malignant lesion, 2 were cases of carcinoma of the colon in which there was no evidence of ulcerative colitis; in 7 others an operation was not performed because other clinical findings did not corroborate the roentgenologic diagnosis; in another 7 cases resection of the colon was performed for chronic ulcerative colitis with what proved to be stricture or polypoid hyperplasia; and in the remaining 4 cases exploration revealed ulcerative colitis but failed to reveal carcimona, and no resection was performed.

In 1949, Sauer and Bargen[4] in a report of 41 cases of carcinoma complicating chronic ulcerative colitis stated that the distribution of carcinomas was as follows: 19 in the rectum, 5 in the sigmoid, 4 in the descending colon, 6 in the splenic flexure, 6 in the transverse colon, 3 in the hepatic flexure, 4 in the ascending colon, and 10 in the cecum. (More than one carcinoma was present in some patients.)

The present review of the roentgenologic manifestations of carcinoma in ulcerative colitis will be concerned only with neoplasms occurring above the level of the rectosigmoid, since lesions in the rectum may be more easily and accurately demonstrated by proctoscopy. The distribution of carcinoma was as follows: transverse colon, 14; splenic flexure, 4; hepatic flexure, 2; descending colon, 7; ascending colon, 6; cecum, 3; sigmoid, 1. Of interest is the relatively large number of occurrences in the transverse colon as well as the solitary case in the sigmoid. Multiple lesions were present in some cases. In all but 2 of the 29 cases the entire colon was involved with ulcerative colitis. When the carcinoma occurred in segmental ulcerative colitis, as it did in the remaining 2 cases, it showed no features different from those of the carcinoma in the other 27 cases.

CLINICAL OBSERVATIONS

It was pointed out in a previous article by one of us[4] that chronic ulcerative colitis began in early adult life in the majority of patients in whom associated carcinoma developed. Few patients were more than thirty-five years of age when symptoms began. In most instances these patients tended to do fairly well on treatment for a time, but they usually had rather severe

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exacerbations during the course of their illness.

Most patients noted a change in the character of their symptoms which tended to point to the fact that a complicating malignant lesion was developing or was present. All had had ulcerative colitis a number of years before the change in the character of the symptoms occurred. The outstanding new symptoms consisted of progressive loss of weight, abdominal pains or cramps differing from the distress usually associated with colitis, an increase in the number of stools, and an increase in the appearance of blood in the stools. In some instances an abdominal mass was noted by the patient. Practically all patients complained of weakness. Some presented signs and symptoms of intestinal obstruction. Hypochromic anemia of rather severe degree was a fairly constant finding in association with the malignant change.

In the present group of 29 cases the average age at the time of discovery of the carcinoma was thirty-five years; the youngest patient was twenty-eight, the oldest sixty-two. The number of years that chronic ulcerative colitis had been present averaged seventeen. The shortest history of the disease was one year and the longest thirty-nine years.

ROENTGENOLOGIC FEATURES

The form carcinoma may take in chronic idiopathic ulcerative colitis may vary considerably. In general, five morphologic groups may be recognized roentgenologically: (1) constricting, (2) annular, (3) sessile, (4) polypoid, and (5) scirrhous. The term "scirrhous" actually does not indicate form, but the pathologist uses this term to describe a type of carcinoma occurring in ulcerative colitis, and this type has a fairly specific roentgenologic picture.

The constricting lesion is characteristically long. In 1 case such a carcinoma was $8\frac{1}{2}$ inches long (Fig. 1). This is the most commonly encountered form of carcinoma in ulcerative colitis and represents more than half the total number of carcinomas in this series. The edges of the bowel involved by carcinoma are ragged and irregular. The lumen of the bowel is narrowed, and its ragged and irregular channel contains numerous small, polyp-like projections (Fig. 2). The ends of the constricted segments are cone-shaped, and they contain an occasional sharp edge of advancing carcinoma. The constriction may be concentric or eccentric, but it is always irregular. There is usually no sharp delineation between carcinoma and adjacent mucosa involved with ulcerative colitis.

The annular lesions resemble the napkin-ring carcinomas of the colon seen in patients without chronic ulcerative colitis. These lesions tend to be shorter, the ends are sharper, and the mucous membrane has the structureless mucosal relief pattern of carcinoma (Fig. 3).

Sessile carcinomas (Fig. 4) are also similar to those encountered in patients without ulcerative colitis. Annular and sessile carcinomas represented 6 of the 37 carcinomas seen in this group of 29 cases. (Some of the patients had multiple carcinomas.) Polypoid carcinomas were un-

FIG. 1. Long carcinoma, involving the cecum, ascending colon, and transverse colon.

Fig. 2. Long carcinoma of the ascending colon, with multiple small polypoid defects in the lumen.

usual in this series; 4 pediculated polypoid carcinomas were observed. This form of carcinoma associated with ulcerative colitis may be more commonly seen by the proctologist.

So-called scirrhous carcinoma (Fig. 5) results in smooth, narrowed segments of colon without polypoid hyperplasia and without the ragged edges or irregular channel that is seen with the constricting lesions. In scirrhous carcinoma there may be no clinical or roentgenologic suspicion of neoplasm; these carcinomas may represent

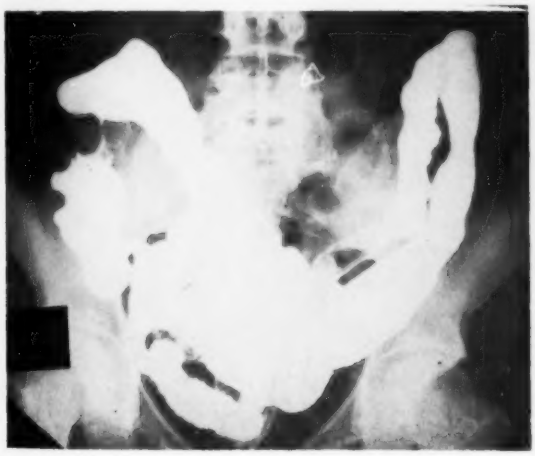

Fig. 3. Annular carcinoma in chronic ulcerative colitis.

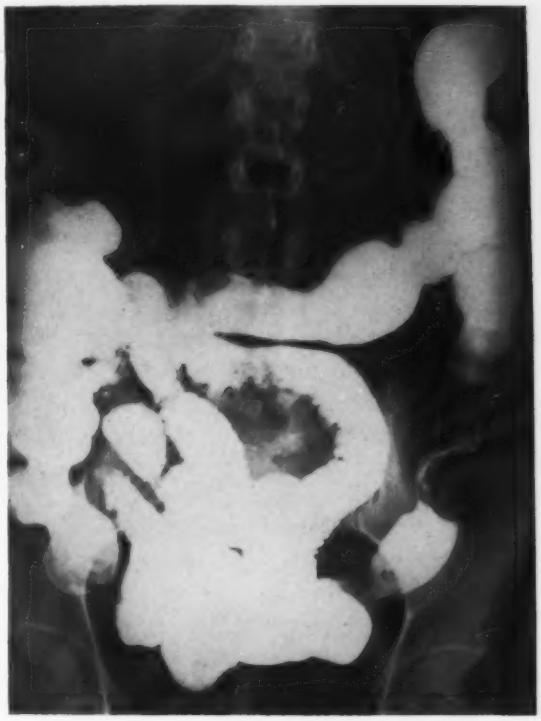

Fig. 4. Sessile carcinoma of the descending colon associated with chronic ulcerative colitis.

Fig. 5. Scirrhous carcinoma of the descending colon. Roentgenologically this is difficult to distinguish from stricture.

the "surprise cancer" found only at resection. Because of replacement of the mucosa by sheets of cancer cells, the bowel has a relatively flat surface which is indistinguishable roentgenologically from the denuded mucosa of burned-out chronic ulcerative colitis. When some segmental narrowing of the bowel is present, differentiation between carcinoma and stricture is difficult, since the flat surface of the lumen of the narrowed segment of bowel may look exactly the same in both scirrhous carcinoma and stricture.

In about half of our cases of carcinoma of the colon in chronic ulcerative colitis, the colitis was quiescent at the time of diagnosis of the carcinoma, and in most of the remaining cases only slight roentgenologic evidence of activity was present. However, carcinoma is not necessarily limited to those patients with contracted, nondistensible, hoselike colons (Fig. 6, *a* and *b*). Many patients with old, burned-out ulcerative colitis have some degree of distensibility of the colon. Distention of the bowel may reveal otherwise unsuspected lesions. For this reason fluoroscopy and roentgenograms of the barium-filled colon are important in the diagnosis of carcinoma.

Although obstruction did not occur in the present cases of carcinoma, it undoubtedly occurs occasionally in carcinoma associated with ulcerative colitis. The 2 cases of complete obstruction roentgenologically thought to involve carcinoma proved to represent strictures with no evidence of carcinoma. It may be that the scarring caused by ulcerative colitis prevents obstruction until the carcinoma is far advanced. The diagnosis then will usually have been made before obstruction occurs. In general, the lumen of the bowel at the region of carcinomatous growth is larger than that seen in carcinomas not associated with chronic ulcerative colitis.

In most cases the usual sharp delineation between carcinoma and normal bowel is not present. The margins of the carcinoma tend to fade off into the adjacent bowel already deformed by ulcerative colitis.

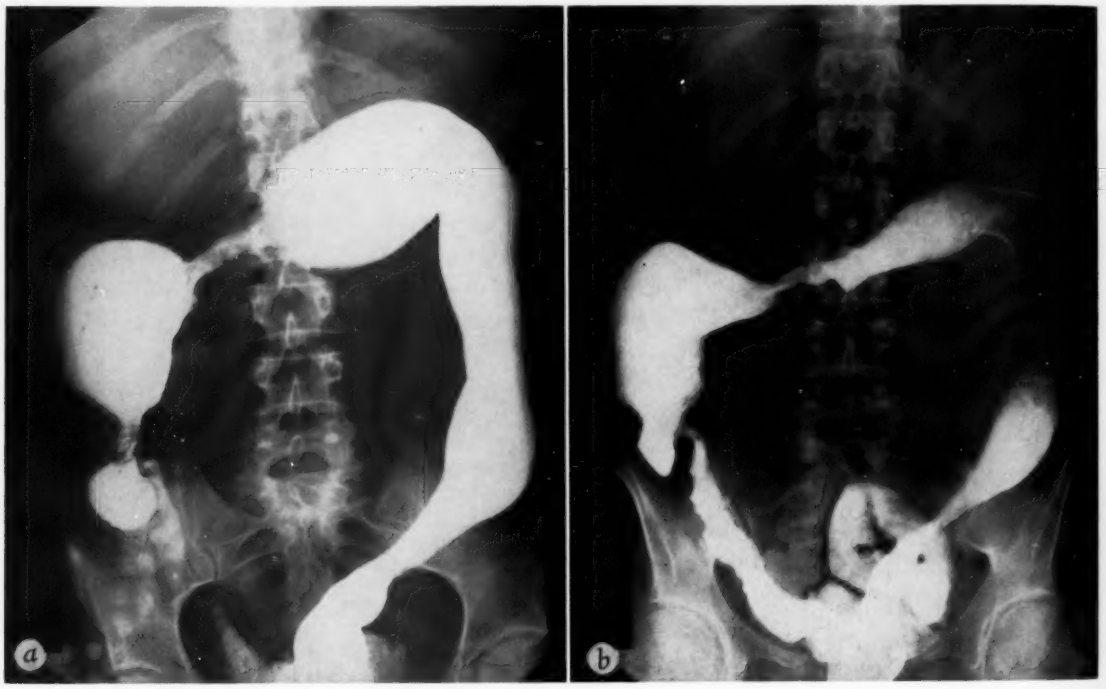

FIG. 6. (*a*) Carcinoma of the transverse colon. Note that distensibility of the colon is still present in this case of ulcerative colitis. (*b*) Postevacuation roentgenogram of *a*.

This is particularly true of the scirrhous variety of carcinoma.

Multiplicity of carcinomas is a fairly common finding in ulcerative colitis. Of the 29 patients in this series, 7 had multiple carcinomas. In most instances these multiple carcinomas were of the same morphologic type, although all different types may occur in the same patient. A higher incidence of multiplicity of carcinoma has been reported in ulcerative colitis,[4] but it must be considered that rectal lesions were excluded in the present study.

Roentgenologic evidence of activity of the ulcerative colitis is unusual. In the majority of instances the disease is quiescent. In about 25 per cent of cases carcinoma is associated with roentgenologic evidence of mild or moderate activity of the colitis. Conflicting opinion exists concerning the relationship of pseudopolypoid hyperplasia, polypoid hyperplasia, and the development of carcinoma in ulcerative colitis. The findings in the present series of 29 patients indicate that polypoid hyperplasia of the colon is less likely to be present in association with carcinoma if it is not localized in a segment of colon.

Fig. 7. Polypoid hyperplasia of the descending colon and sigmoid. No carcinoma is present.

Fig. 8. Strictures (arrows) in the terminal ileum near the ileocecal valve and also in the descending colon. Carcinoma (scirrhous type) is present in the sigmoid.

DIFFERENTIAL DIAGNOSIS

The most common form of carcinoma associated with chronic ulcerative colitis—the long constricting carcinoma—must be distinguished from localized polypoid hyperplasia of the colon (Fig. 7). In general, the carcinoma will produce more ragged edges, a more irregular channel, and more constricting deformity. Because the changes differ only in degree, the differentiation between segmental polypoid hyperplasia and carcinoma may be difficult. However, none of the cases of simple polypoid hyperplasia showed the ragged, irregular destruction characteristic of carcinoma. With this type of lesion, diagnosis usually involves careful appraisal of the clinical status of the patient as well as roentgenologic evaluation. Often a definite diagnosis can be made only after surgical resection.

Annular, sessile, and polypoid lesions offer no serious problem in diagnosis, and they should be removed.

There seems to be no reliable way of determining the difference between benign stricture and so-called scirrhous carcinoma associated with chronic ulcerative colitis (Fig. 8). Close attention to the clinical status of the patient may be of some help. Exploration may be necessary for differentiation.

SUMMARY

The occurrence of carcinoma superimposed on ulcerative colitis is a well-established fact. Carcinoma tends to occur in younger persons with quiescent ulcerative colitis who have a history of the disease of about seventeen years' duration. In such cases the patient may complain of a recent change in bowel symptoms.

Roentgenologically, these lesions consist of constricting, sessile, polypoid, annular, and scirrhous defects. The most common form is the long constricting lesion with coned ends, ragged edges, small polyp-like defects in the lumen, and an adequate but ragged channel.

Differential diagnosis is difficult, particularly in the scirrhous variety, in which the lesions may often resemble strictures roentgenologically. Occasionally, resection of the colon will reveal a carcinoma which was not suspected clinically or roentgenologically.

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CARCINOID OF THE RECTUM*

A CASE REPORT WITH OBSERVATIONS ON RADIOSENSITIVITY OF NODULAR METASTASES TO THE SKIN

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CARCINOIDS are said to arise from argentaffin (Kultschitzky) cells normally present in the bases of the crypts of Lieberkühn in the gastrointestinal tract from the cardia of the stomach to the anus. Seventy to 90 per cent of the cases occur in the appendix. Most of the others are found in the ileum. The rectum is probably the most frequent site of origin in the colon. Multiple primary lesions are not uncommon, particularly in the small intestine. They have been found in patients ten days to eighty-nine years of age. Statistical data are summarized by Ritchie.[13] De Lashmutt and Kolozsi[3] found that the average age of patients with this tumor was forty-five years. Green[7] reports that 3 of his 6 cases ranged from twenty-one to thirty-two years in age.

Except for the carcinoids arising in the appendix where symptoms resembling the initial stages of appendicitis lead to early discovery, metastases are often quite large before a small primary lesion is found.[11] The metastatic spread occurs through the lymphatics of the mesentery to the regional lymph nodes and by the portal circulation to the liver. Metastases also have been reported in the bone, brain, skin and viscera. In 1944, in a survey of the literature, Ritchie and Stafford[14] found that only 12.6 per cent of 111 cases of rectal carcinoids had metastasized. This was the lowest incidence save for carcinoids of the appendix. These figures would have undoubtedly been revised upward with adequate follow-up. Survival for more than ten years is not uncommon and metastases from the rectum which were not grossly evident on an earlier observation may well appear later. Dockerty[4] states that nearly 50 per cent of rectal lesions larger than 5 mm. when first seen will metastasize. He also states that 5 per cent of submucosal nodules in the rectum seen by the proctoscopist are carcinoids. They are usually small, freely movable, and respond to fulguration. About 75 per cent of them are asymptomatic and are found incidentally. Symptomatic patients may have bleeding, diarrhea, constipation, and tenesmus.

Masson, forty years ago, referred to these tumors as argentaffinomas and insisted on the endocrine activity of the enterochromaffin cells. This was regarded as unfounded speculation. Since 1930, case reports appeared in the literature noting the coincidence of pulmonary stenosis and metastasizing carcinoid, but it was not until 1953 that two independent reports[9,15] established a new syndrome linking hormonal effects and cardiovascular abnormalities with the tumor. Since then other cases have been published.[10,18] In 1936, Erspamer[5] and his co-workers extracted a substance, which he called "enteramine" from tissues containing Kultschitzky or similar chromaffin cells. In 1952, this was identified as 5-hydroxytryptamine by Erspamer and Asero.[6] Page called this "serotonin."[12] It is now evident that some carcinoid tumors produce an excess amount of this substance causing violaceous cyanosis and telangiectasia of the face, episodic flushing, and at times tachycardia, hypotension, asthma, diarrhea, and abdominal pain. As it passes through the right heart, it apparently has a toxic effect on the endo-

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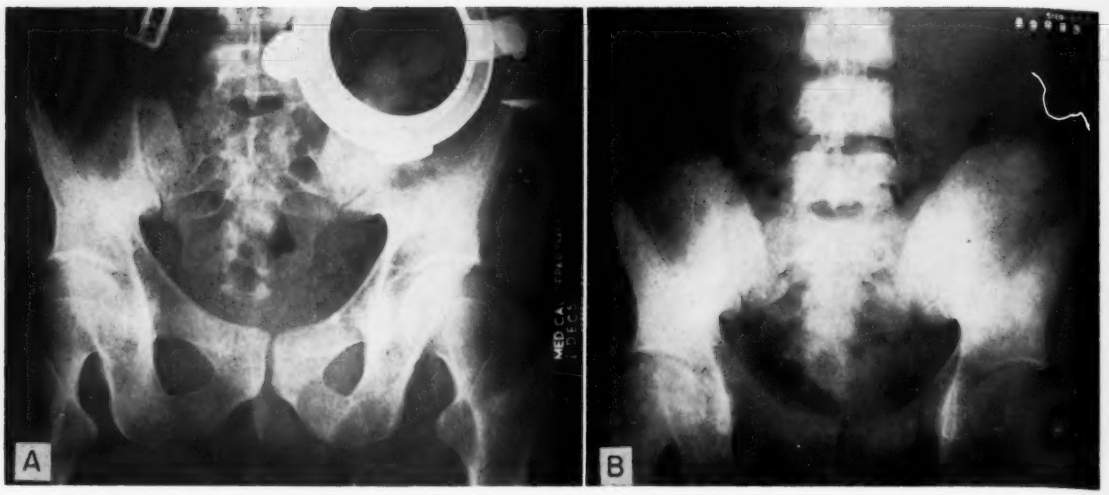

Fig. 1. Roentgenograms of the pelvis. (*A*) December, 1953. Early evidence of osteoblastic metastases was manifested by dense irregular trabeculations. These are seen in the pelvis. Note the colostomy. (*B*) April, 1956. The osteoblastic metastases progressed to dense homogeneous involvement of the lumbar spine and most of the pelvis. The process is less advanced in the pubic arches and upper femora. These changes were typical for the entire axial skeleton except for the skull which showed no metastatic lesions.

cardium, resulting in right heart failure from a fibrous endocarditis and valvulitis of either or both the pulmonic and tricuspid valves. For the most part, serotonin is carried by the platelets and is apparently inactivated by the lungs, sparing the left heart. It is excreted in the urine as 5-hydroxyindole-acetic acid.

REPORT OF A CASE

G.B. In October, 1948, this thirty-eight year old white male truck driver had three episodes of bloody bowel movements with a total loss of about a pint of dark red blood. Three weeks later he again had three bowel movements mixed with blood. A private physician found a small rectal lesion on a sigmoidoscopy. This was biopsied and reported as carcinoma.

The patient was referred to Oak Knoll Naval Hospital in Oakland in December. A 1 cm. papilloma on the anterior rectal wall 10 cm. from the anus was visualized through the sigmoidoscope. A biopsy was reported as carcinoid. An abdominoperineal resection was done on December 28, 1948. The rectum and sigmoid were removed. No gross lymphadenopathy was observed and the liver was normal to palpation. An ulcerated lesion, 17 mm. in diameter, with an elevated border was found in the rectum. Sections did not indicate any invasion of the muscularis. The pathologist was uncertain of the histology but thought it was a melanosarcoma. He noted that no chromaffin cells were found on special stains. It was reported as a malignancy, type undetermined. The patient was discharged feeling well with a functioning left lower quadrant colostomy. He returned to his former occupation and had no complaints until August, 1953 when he began to lose weight, became easily fatigued, and noted anorexia, as well as back and leg pain. Roentgenograms in December, 1953 showed dense irregular trabeculations in the lumbar spine and pelvis (Fig. 1*A*).

He was first seen in this hospital in March, 1954 and, at that time, was thoroughly investigated. It was felt that the patient had widespread metastatic bone disease (Fig. 2*A*). A biopsy of a skin nodule on the face revealed metastatic carcinoid. Bone marrow biopsies at that time were negative. Severe low back pain began in October, 1954. At that time the alkaline phosphatase was elevated to 16.1 King-Armstrong units. The hemoglobin was 12 gm. and the white blood cell count was 9,500 with a normal differential. Thioguanine was started in November and continued for eighteen months in the hope of arresting the growth of neoplasm. Initially, the dose was 120 mg. per day but, because of bone marrow depression requiring several transfusions, it was gradually

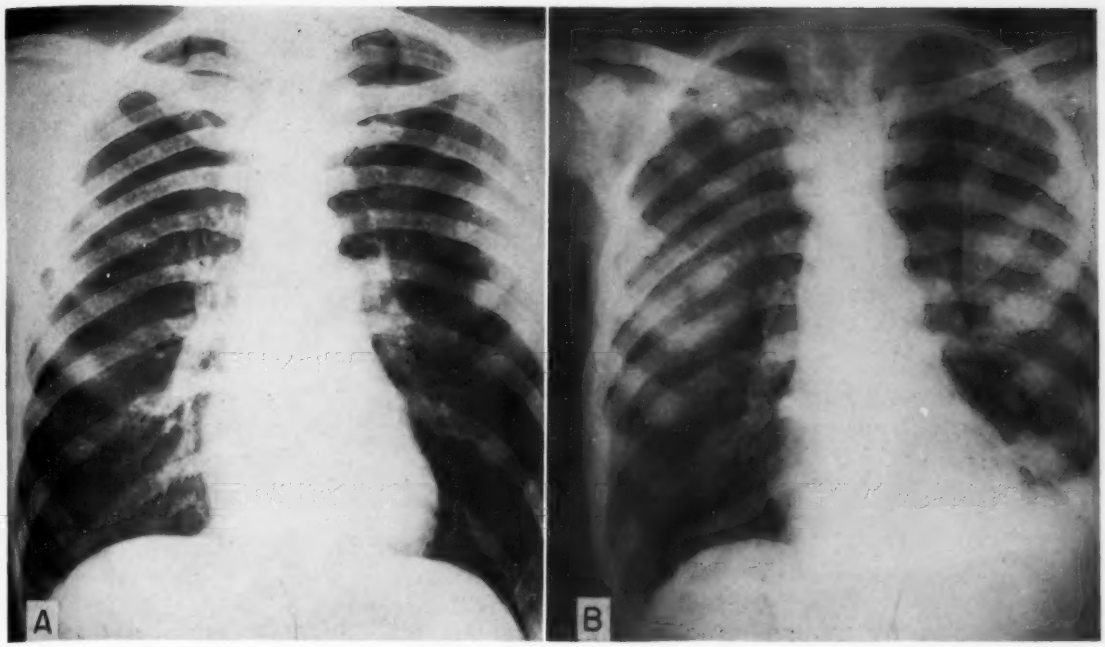

Fig. 2. Roentgenograms of the chest. (*A*) April, 1954. The osteoblastic process had diffusely infiltrated the ribs and thickened the trabeculae. Similar changes were found in the spine and pelvis. (*B*) April, 1956. The density of the ribs had increased markedly, and many of them are expanded. Fluid had collected at the left base.

reduced to 75 mg. per day. There was a gradual downhill course noted through several interval admissions. The diffuse osteoblastic metastases progressed unabated. Bone pain required continuous use of narcotics. Increasing numbers of skin nodules, up to 15 mm. in diameter, appeared on the face, scalp, arms and trunk. Some of these caused discomfort and were excised, confirming the diagnosis of metastatic carcinoid.

The final admission was in April, 1956 for edema, ataxia, numbness of the lower extremities and unremitting severe low back pain. The liver was four fingerbreadths below the costal margin. Thioguanine was discontinued since it was obviously ineffective. During this hospital stay there was increasing paresis, rising to the midabdomen. Bilateral Babinski signs appeared, and there was weakness of the small muscles of the hand. Clinically, it was felt that the patient had multiple metastases to the lumbar and cervical cord, involvement of the cauda equina and the lumbosacral plexus, and venous obstruction. A myelogram was unsuccessful. Roentgenograms of the spine, pelvis, humeri, femora and chest showed continued progression of the osteoblastic metastases (Fig. 1*B* and 2*B*). No metastases were visible in the skull. Fluid had collected at the left lung base. Laboratory data were as follows: spinal fluid slightly xanthochromic with 101 red blood cells, 1 lymphocyte, and 496 mg. per cent total protein; urine negative for melanin and serotonin; total serum protein 5.3 gm. per cent, albumin 3.0 gm. per cent, globulin 2.3 gm. per cent. The complete blood cell count on June 28 was hemoglobin 6.1 gm., white blood cells 4,450, differential normal, platelets 37,000, hematocrit 22.5.

He was referred to the radiologic department for palliative irradiation of the lumbosacral region for relief of back pain, venous obstruction and nerve root compression. A review of the literature gave no information regarding the radiosensitivity of this neoplasm, other than that it responded poorly to irradiation. Radiation therapy was begun on May 17 and continued for twenty-four days. A tumor dose of 2,100 r (half value layer 3 mm. Cu) was administered at a depth of 6 cm. The air dose was 2,380 r, and the skin dose was 3,150 r. No significant relief of pain was achieved and there

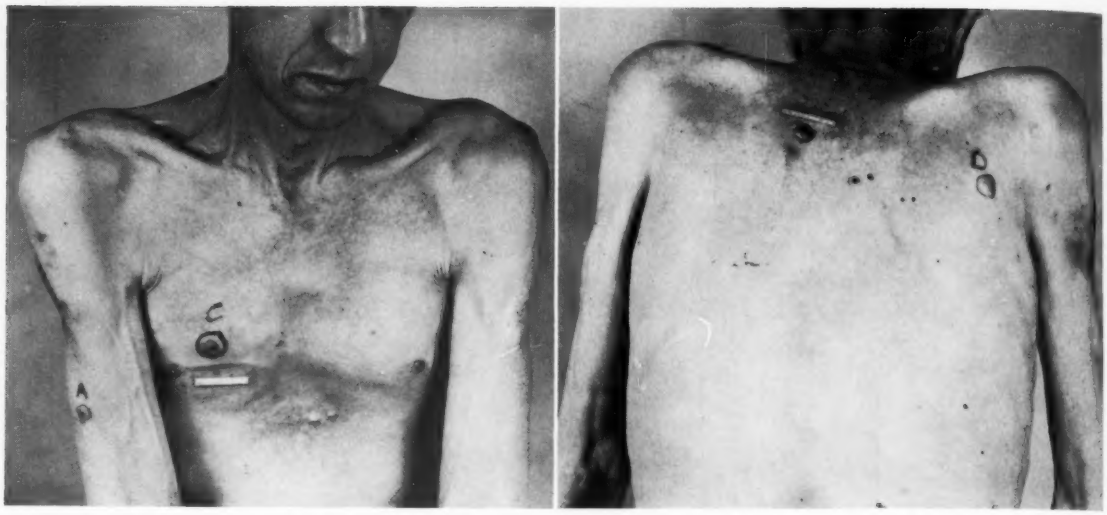

Fig. 3. May, 1956. Multiple skin nodules up to 15 mm. in diameter.

was no effect on the venous obstruction or on the neurologic changes.

A time dose study was undertaken on some of the nodules (Fig. 3) to determine the disappearance dose of irradiation, but this had to be abandoned shortly after it was under way because the deteriorating condition of the patient did not permit its continuation. The data collected are shown in Table 1 and Figure 4.

The first convulsion occurred on June 11. This was accompanied by apraxia and apnea, indicating cerebral metastases. Dilantin was started but this did not prevent recurrence. The patient continued to decline and expired on July 2, 1956, after an eight year history. No autopsy was obtained.

DISCUSSION

Dockerty's[4] comment regarding the high incidence of metastases from rectal lesions larger than 5 mm. was applicable in this case. About three weeks before the abdominoperineal resection, the lesion was a 1 cm. superficial papilloma. Although no metastases were found at surgery or in the specimen, five years later there was clinical evidence of this, confirmed by biopsy of skin nodules and roentgenographic evidence of diffuse metastases to bone. The dense homogeneous character of the osteoblastic process without a lytic component progressing from thickened, somewhat irregular, trabeculae is indeed interesting and attests to a slow stimulation of bone growth by the tumor. No serotonin was observed.

There is no definitive information in the literature regarding the radiosensitivity of this tumor. The multiple skin nodules provided an excellent opportunity for time dose studies but, unfortunately, the terminal status of the patient did not allow us to obtain any useful data for more than six days. Most of the observations in Table 1 and Figure 4 are for a one day period. The

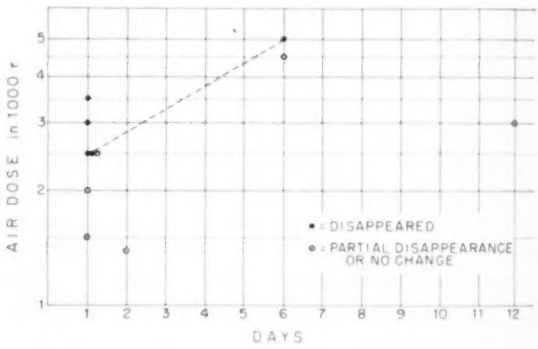

Fig. 4. Observation on the radiosensitivity of the skin metastases. The minimum disappearance dose for one treatment was 2,500 r in air. The data are inadequate for fractional doses. The minimum disappearance dose for six days may be 5,000 r in air.

TABLE I

OBSERVATIONS ON RADIOSENSITIVITY OF SKIN METASTASES

Nodule	Location	Lesion Diameter (mm.)	Field Diameter (mm.)	Half Value Layer (mm. Cu)	Fractions	Air Dose per Fraction	Total Treatment Time (days)	Total Air Dose	Last Observation after Final Treatment (days)	Skin Erythema	Comment
A	right arm	7.5	10	1.72	2	500	3	1,000	41	None	no change
B	posterior thorax	10	15	1.72	3	1,000	12	3,000	35	moderate 1°	no change
C	right pectoral	11	15	1.72	1	1,500	1	1,500	47	None	no change
D	posterior thorax	12	15	1.72	1	2,000	1	2,000	24	Moderate 2°	no change
E	posterior thorax	15	15	1.72	2	710	2	1,420	41	moderate 1°	no change
F	left shoulder	15	15	1.72	2	570	7	1,040	35	mild 1°	no change
G	posterior cervical region	8	10	1.72	1	2,500	1	2,500	33	Mild 1°	partial regression
H	cervical region	10	20	.57	1	2,500	1	2,500	28	moderate 2°	disappeared
I	posterior thorax	7	20	.57	1	3,000	1	3,000	28	mild 1°	disappeared
J	abdomen	7	20	.57	4	1,250	6	5,000	23	moderately severe 1°	disappeared
K	abdomen	10	20	.57	4	1,125	6	4,500	23	moderately severe 1°	small residual
L	sternum	5	20	.57	1	2,500	1	2,500	27	moderate 2°	disappeared
M	left arm	5	20	.57	1	3,000	1	3,000	25	moderate 1°	disappeared
N	left lumbar region	10	20	.57	1	3,500	1	3,500	23	moderately severe 1°	disappeared

minimum disappearance dose of 2,500 r for one day and 5,000 r for six days indicates that carcinoid is at least as radioresistant as squamous cell carcinoma and probably as resistant as adenocarcinoma. Cure, therefore, of visceral tumors by irradiation cannot be expected, even at the hazard of doing irreparable damage to normal tissues.

SUMMARY

A case of carcinoid of the rectum is presented. The patient was asymptomatic for five years following an abdominoperineal resection after which there were widespread metastases to bone, skin, viscera, spinal cord, and brain, respectively, with a fatal termination after eight years. Interesting features were dense osteoblastic metastases to the axial skeleton (skull excepted) and multiple skin nodules. Radiosensitivity studies on some of the nodules demonstrated a minimum disappearance dose of 2,500 r in air for one day and 5,000 r in air for six days.

The literature for carcinoids is reviewed briefly with particular reference to rectal lesions and to interesting hormonal effects exhibited by some of these tumors.

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MESENTERIC ARTERIOGRAPHY*

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THERE has been little basic progress in the roentgenography of the gastrointestinal tract since Dr. Cannon introduced radiopaque contrast material into the gut of a cat. Although refinements of his basic methods have been made, all deductions as to the existence of an abnormality in the gastrointestinal tract roentgenographically still are dependent upon the detection of the lesion's effect on the lumen of the gut. Although dependable for demonstrating the shape and contour of the inner surface of the bowel, existing intestinal roentgenography fails to provide means of assessing the architecture and limits of the whole lesion. Consequently, roentgenologic inferences as to the specific cellular characteristics of a given lesion of the intestine can be little more than an intelligent guess based largely upon frequencies. This applies especially to the problem of differentiating benign from malignant ulcers of the stomach and benign polyps from polypoid carcinomas in the stomach and colon. In an attempt to learn more about the roentgenographic anatomic details of intestinal lesions, both neoplastic and inflammatory, we have been employing operative and isolated organ mesenteric arteriography.

Bierman *et al.*[1] succeeded in opacifying the celiac axis, the superior and inferior mesenteric arteries by transaortic intra-arterial catheterization of viscera. The procedure was unpredictable and dangerous. Ödman[5,6] visualized the superior mesenteric artery by transfemoral arteriography. The results of this procedure are difficult to interpret because of superimposition of the opacified vessels of the intestinal mesentery.

Schobinger, Blackman and Lin[7] and Schobinger[8] demonstrated vascular patterns of benign and malignant neoplasms of the colon and of diverticulitis.

This study was undertaken to develop and to evaluate intestinal arteriography as a method for differentiating neoplastic from inflammatory lesions of the gastrointestinal tract and for distinguishing benign from malignant neoplasms.

METHOD

A limited trial of Schobinger and his coworkers'[7] method was disappointing because of the insufficient vascular detail obtained with it. In order to achieve the necessary detailed opacification of the intestinal vessels, we have used a film cassette wrapped in sterile plastic, placing it directly under the bowel. Then the roentgenograms were made before and during the terminal period of the injection of 8 to 10 ml. of contrast material into the proper artery through a plastic catheter or needle. The contrast material we first used was a solution of 37.5 per cent of diacetamido-triiodobenzoate (hypaque). However after Grayson *et al.*[2] showed that 40 per cent methylglucamine diacetylamino-triiodobenzoate (renografin) was less toxic to the bowel than hypaque, we used renografin exclusively. In order to facilitate subsequent arteriography of the removed piece of gastrointestinal tract, the plastic catheter was left in the proper artery after the *in vivo* examination was done.

CASE MATERIAL

Mesenteric arteriography was performed during an operation 35 times and upon 48

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TABLE I

LESIONS STUDIED BY MESENTERIC ARTERIOGRAPHY

		Lesion	No.
Operative Arteriography	Colon	Carcinoma	15
		Adenomatous polyps	6
		Villous adenoma	1
		Lipoma	1
		Mucocele of appendix	1
		Chronic ulcerative colitis	3
		Diverticulitis	1
		Multiple small arteriovenous malformations	1
	Stomach	Carcinoma	3
		Peptic ulcer	3
		Total	35
Arteriography on Specimens	Colon	Carcinoma	28
		Adenomatous polyps	2
		Lipoma	1
		Villous adenoma	1
		Chronic ulcerative colitis	6
	Stomach	Carcinoma	6
		Leiomyosarcoma	1
		Peptic ulcer	3*
		Total	48

* One also had antral gastritis.

stomachs or colons after their removal from the body during celiotomy (Table I).

RESULTS

The operative arteriograms of the intact bowel usually showed better arterial opacification than did the isolated organ arteriograms and therefore were easier to interpret. The vascular pattern outlined in an organ *in vivo* did not differ significantly from the pattern observed after the same organ was isolated.

CARCINOMA OF THE COLON

In all instances, operative and resected specimen arteriography of colonic carcinoma demonstrated destruction of the normal arcades of intestinal vessels. These were replaced by abnormal channels which were tortuous, angulated, had an irregular lumen, and coursed through the lesion chaotically in many directions (Fig. 1, *A–D;* and 2, *A* and *B*). The irregularity of lumen consisted of sudden irregular widenings and narrowings. In many roentgenograms the vessels of the tumor were finer and narrower than those of the adjacent intestine. The arteriograms of all colonic carcinomas contained irregular, undelimited areas of opacification. These were either small patchy areas of radiodensity resembling lakes surrounding vessels or large uniform areas of laking in which discrete vessels could not be seen. The small perivascular lakes were seen on all the arteriograms of colonic cancer. The larger areas of laking were found in 8 of 15 carcinomas injected *in vivo* and in 8 of the 18 tumors studied following their removal. These areas of laking were not seen when

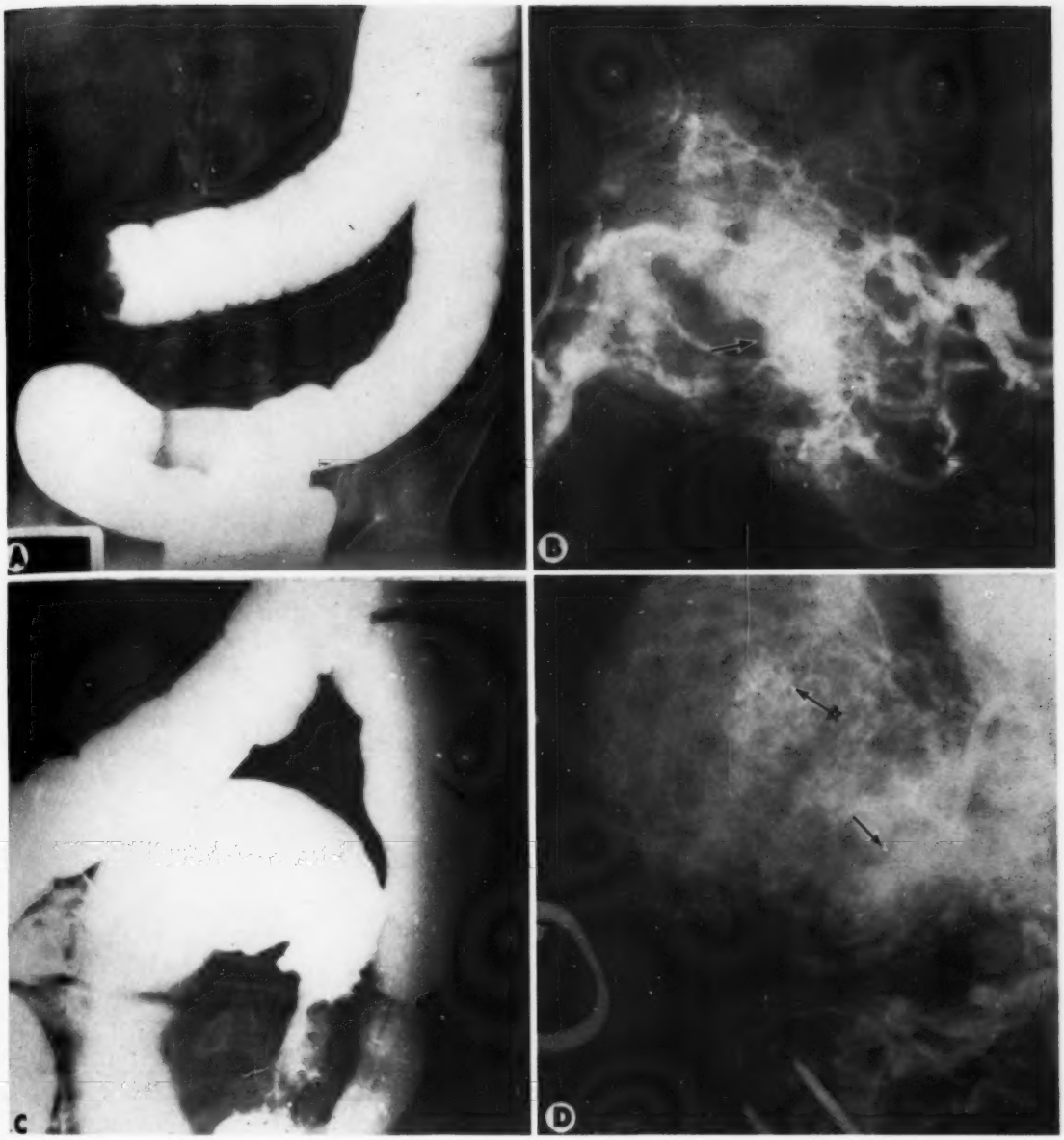

Fig. 1. (*A* and *B*) Annular carcinoma of the transverse colon. (*A*) A pre-evacuation barium enema roentgenogram showing the lesion. (*B*) An operative mesenteric arteriogram showing the lesion. The vessels are irregular and show sharp turnings. The blood vessels, in general, are more numerous than in the surrounding areas; however, close to the top of the lesion there are fewer vessels. Areas of dense opacification are seen in the tumor (arrow). (*C* and *D*) Carcinoma of the sigmoid colon. (*C*) Barium enema roentgenogram. (*D*) A mesenteric arteriogram, *in vivo*. Irregular, chaotically arranged vessels are seen throughout the tumor. There are areas of dense opacification (arrow) as well as smaller multiple perivascular areas of laking (arrow with star).

barium sulfate (USP) was used as the arteriographic contrast material in specimens.

The mesenteric arteries to the tumor always were prominent and numerous, often larger than adjacent mesenteric vessels. They originated from the marginal artery of the mesentery at irregular inter-

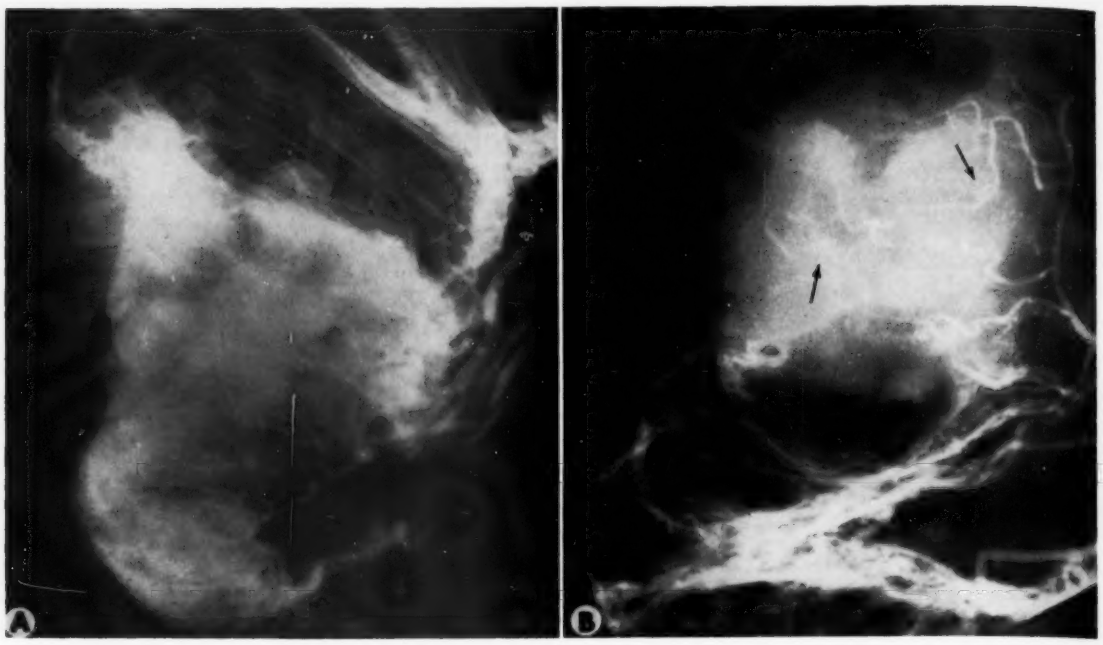

FIG. 2. (*A*) An operative mesenteric arteriogram showing a carcinoma of the cecum. Note that the mesenteric vessels to the tumor are quite large. The tumor shows large areas of laking along with multiple irregular vessels, sharply contrasting with the parallel normal vessels seen in the uninvolved bowel at the top of the figure. (*B*) Relatively avascular carcinoma of the sigmoid. The vessels in the tumor show an irregular lumen, sharp turnings and, in general, a chaotic direction. Arrows point to small areas of perivascular laking.

vals and often showed dilatations resembling aneurysms. The vascular channels visualized within the tumors were of smaller caliber and number than in the normal bowel wall in 3 of 15 cancers. Only 2 of the 15 carcinomas studied *in vivo* exhibited vessels uniformly more numerous than normal. The remaining 10 had areas in which only a few vessels were seen and other areas with numerous abnormal, opacified vessels.

BENIGN LESIONS OF THE COLON

Mesenteric arteriograms were performed in 6 patients being operated upon for adenomatous polyps. One had familial multiple polyposis. All others had one or more polypi that were larger than 2 cm. in diameter. One patient had a large villous adenoma (Fig. 3, *C* and *D*). The lesions in all 7 patients showed multi-directional vessels of larger caliber and number than normal. Looking with a magnifying glass at individual vessels within polyps on the arteriogram, luminal irregularities and sharp turnings were absent, although the vessels often were tortuous. The polyps did not contain diffuse or perivascular laking seen in cancers. All polyps except for the very small had a greater vascularity than did the normal adjacent intestine (Fig. 3, *A* and *B*).

Operative arteriograms of a mucocele of the appendix (Fig. 4, *A* and *B*) and of a colonic lipoma (Fig. 4, *C* and *D*) showed superficial vessels encircling the lesions but few if any within them. These arteriograms differed markedly from those of benign adenomatous polyps and of carcinoma.

The characteristics described serve as the basis for roentgenographic differentiation of benign from malignant colonic tumors (Fig. 1, *A–D*; 2, *A* and *B;* and 3, *A–D*) (Table II).

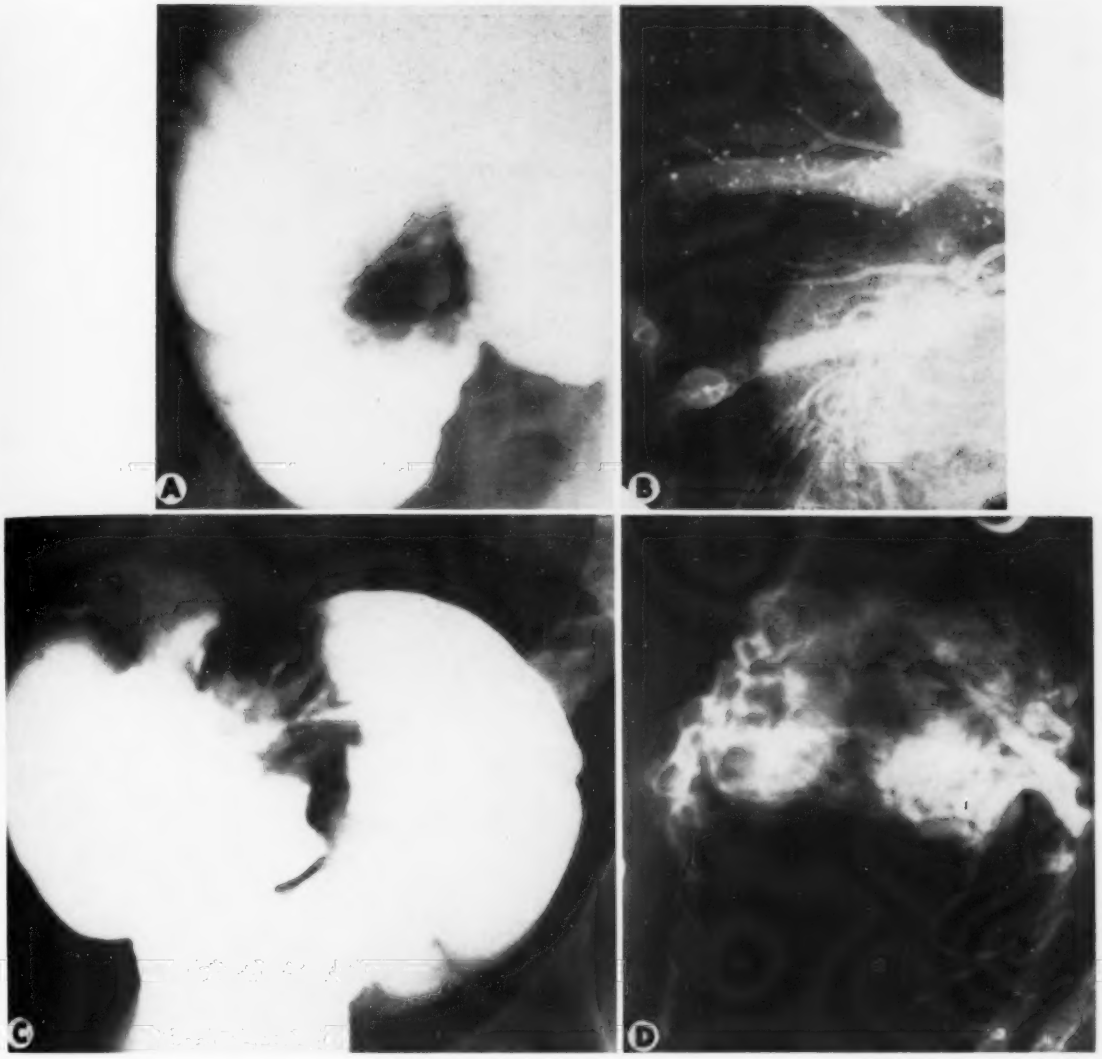

Fig. 3. (*A* and *B*) Adenomatous polyps of the cecum. (*A*) A spot roentgenogram during barium enema study showing the large filling defect in the cecum in the region of the ileocecal valve. (*B*) An operative mesenteric arteriogram showing one very large polyp and one smaller polyp. There are multiple winding vessels within the polyps but no areas of perivascular opacity or large areas of laking. The vessels fail to show irregular lumina and do not show sharp turnings. (*C* and *D*) Villous adenoma of the sigmoid. (*C*) A spot roentgenogram of the sigmoid obtained during a barium enema study. (*D*) An operative mesenteric arteriogram of the lesion showing numerous large tortuous vessels throughout the tumor, sharply contrasted with less numerous and smaller parallel vessels in the adjacent normal bowel. No areas of perivascular laking are seen.

Three patients with chronic ulcerative colitis had operative mesenteric arteriography. Six colons with the same disease were examined by arteriography after their removal. The colonic vessels were numerous, tortuous and gave off branches at close to a 90° angle. The normal parallel vessels forming arcades were replaced by the vessels characteristic of this disease. The difference in appearance, orientation and distribution from vessels in the normal bowel is illustrated in Figure 5*A*. Figure 5*B* is an operative arteriogram of another patient, containing the same type of vessels

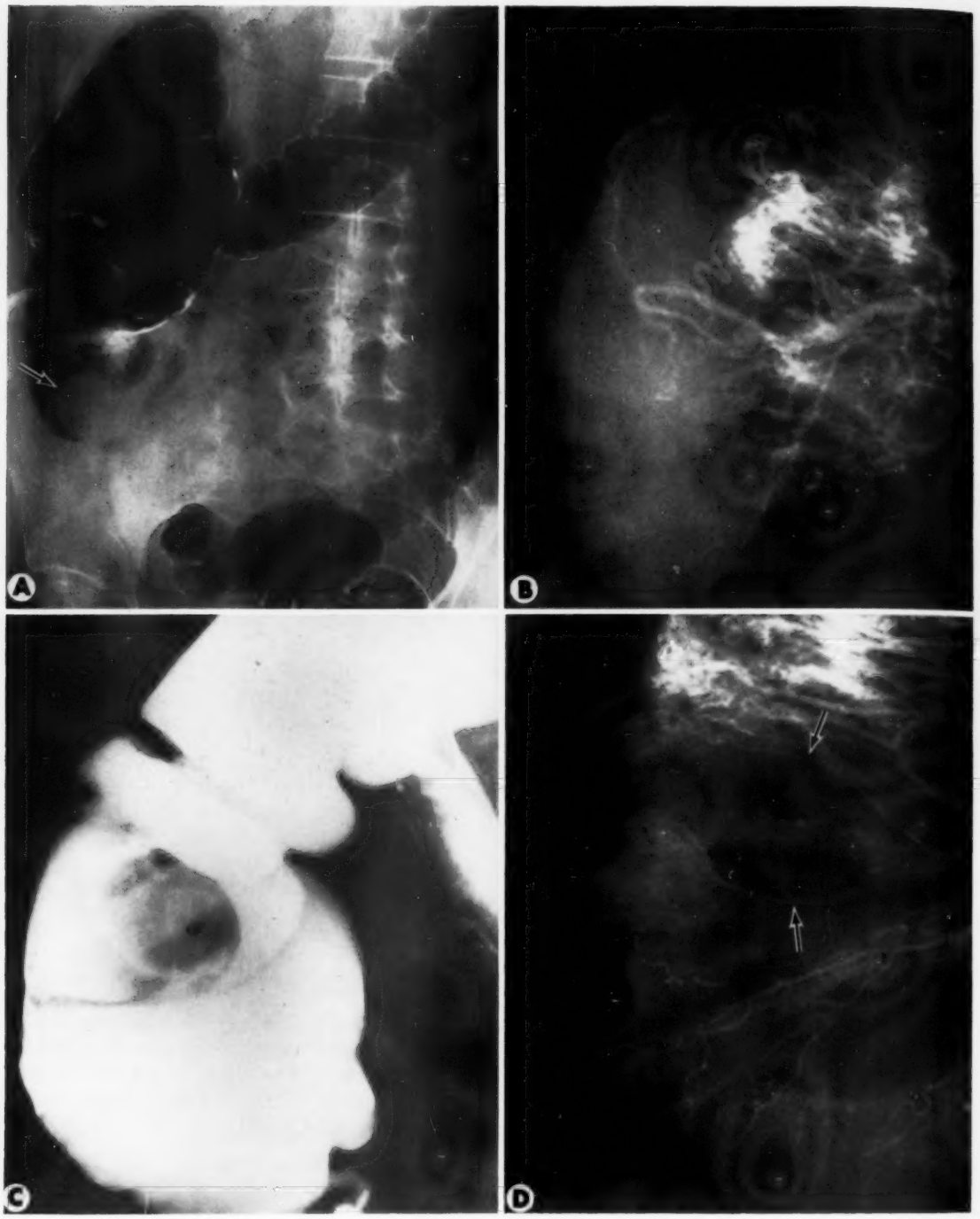

FIG. 4. (*A* and *B*) Mucocele of the appendix. (*A*) A large rounded filling defect is seen on the medial aspect of the cecum (arrow). (*B*) An operative mesenteric arteriogram shows the lesion to be relatively avascular. Only a few vessels are seen coursing on the surface of the tumor, in sharp contrast to the numerous vessels in the mesentery. (*C* and *D*) Lipoma of the cecum. (*C*) A spot roentgenogram of the cecum obtained during barium enema study shows a smooth rounded defect. (*D*) An operative mesenteric arteriogram shows the lesion to be radiolucent and relatively avascular. Only a few vessels are seen coursing on the surface.

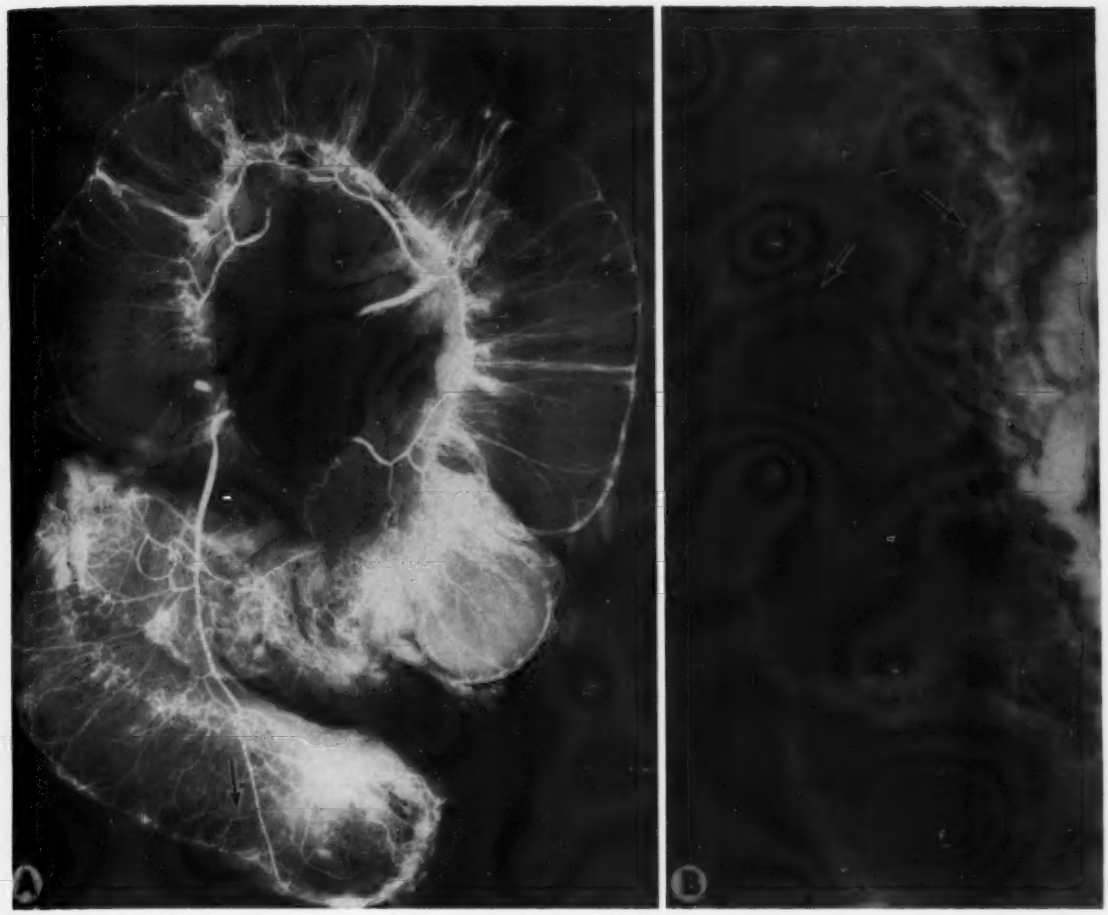

Fig. 5. (*A*) An arteriogram of the operative specimen following total colectomy. The distal colon is severely involved by chronic ulcerative colitis. The bowel wall is greatly thickened. The vessels are more numerous, tortuous and show branching at right angles (arrow). This is in sharp contrast to the normal arrangement of mesenteric vessels in the uninvolved bowel seen on the figure. (*B*) An operative mesenteric arteriogram in another case of chronic ulcerative colitis. Numerous tortuous vessels are seen. Many vessels show branching at right angles.

as seen in the specimen examination. The fecal stream had been diverted for a year in one case. This specimen showed only a few fine parallel blood vessels.

An operative arteriogram of a patient with diverticulitis of the transverse colon showed numerous dilated vessels in the area of inflammation just as seen by Schobinger.[8]

The vascular patterns of chronic ulcerative colitis and diverticulitis were different from that of malignant and benign neoplasms of the colon.

Arteriography of the Stomach

Operative arteriography of the stomach is not a formidable procedure. Anastomoses between arteries are abundant and injection of any vessels of the gastric arcades will visualize the area of abnormality. We have successfully studied lesions of the antrum, body and fundus of the stomach. Arteriograms of polypoid carcinoma of the stomach both *in vivo* and in the specimen showed the same type of vascular pattern described with the more vascular carcinomas of the colon. The normal paral-

TABLE II

FINDINGS IN MESENTERIC ARTERIOGRAPHY

	Lesion	Increased No. of Vessels in Diseased Area	Decreased No. of Vessels in Diseased Area	Areas of Increased and Decreased Vessels Side by Side	Large Areas of Laking	Perivascular Small Areas of Laking	Irregular Vascular Pattern	Vascular Luminal Irregularities	Changes in Directional Coursing of Vessels	Right Angle Branching
Colon	Carcinoma	±	±	Most common	In about 50%*	+*	+	+	+	o
	Adenomatous polyps	+	o	o	o	o	+	o	o	o
	Chronic ulcerative colitis	+	o	−	o	o	o	o	o	+
Stomach	Polypoid carcinoma	+	o	±	+	+	+	+	+	o
	Ulcerating carcinoma	o	+	o	o	o	o	+	o	o
	Peptic ulcer	+	o	±	o	o	o	o	o	o

* Except in specimen injected with USP barium sulfate.

lel arteries were absent. There were areas of diffuse and of small perivascular laking present (Fig. 6, *A–D*; and 7, *A* and *B*). The vessels in the tumor were irregular in lumen and showed sharp turnings in their course. Their direction was chaotic. Although certain areas of the tumor were relatively avascular, the vessels in general were more numerous. Arteries supplying the tumor were larger than those supplying normal areas.

Benign ulcers of the stomach showed numerous, wide, winding vessels surrounding the crater (Fig. 8, *C* and *D*). The immediate location of the crater and its margins may show few or no vessels. The vessels surrounding the ulcer crater were not irregular in either their lumen or distribution. Our series includes two malignant ulcers of the stomach. In both of them no evidence of cancer was detectable in the gross specimen and the cancer cells grew in sheaths surrounded by fibrous tissue. The diagnosis of carcinoma could be made only histologically. The arteriograms of the stomach following removal of the organ showed only a few vessels in the region involved by cancer. No areas of diffuse or perivascular laking could be demonstrated. The vessels appeared finer and smaller than in the arteriograms of benign peptic ulcer. The lumen of some of the vessels showed irregularly spaced, asymmetric areas of narrowing and dilatation. The distribution of the vessels, however, followed the normal pattern (Fig. 8, *A* and *B*). The difference between benign peptic ulcers and ulcerating carcinomas will have to be evaluated by a larger series.

In one case having antral gastritis with a benign peptic ulcer, the arteriogram showed numerous vessels of normal ap-

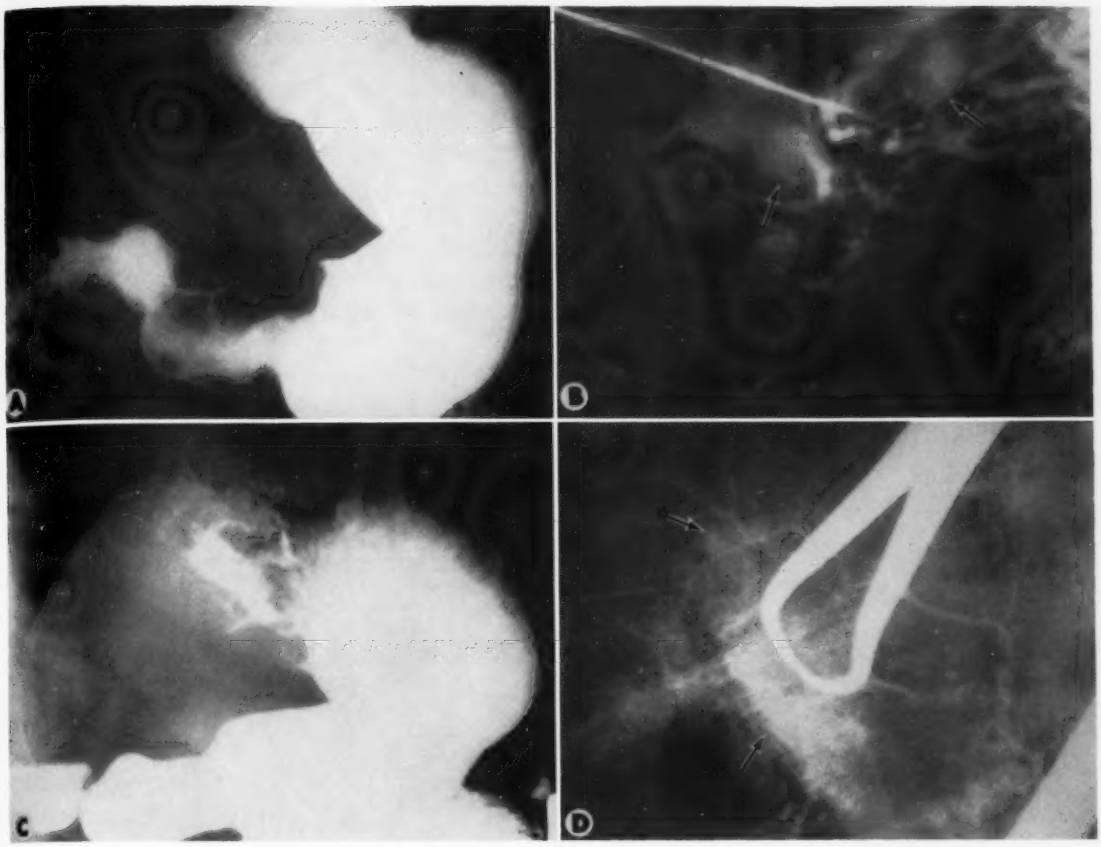

Fig. 6. (*A* and *B*) Polypoid carcinoma of the antrum of the stomach. (*A*) An upper gastrointestinal study with barium. (*B*) A mesenteric arteriogram with injection into the right gastric artery. Arrows point to areas of laking in the tumor. The vessels leading to the tumor are large and numerous. (*C* and *D*) Carcinoma of the fundus of the stomach. (*C*) An upper gastrointestinal study with barium. A large ulcer is seen within the mass in the fundus of the stomach. (*D*) An operative mesenteric arteriogram with injection into the left gastric artery shows areas of increased and decreased vascularity side by side. The vessels are irregular in distribution. Large areas of opacity in the tumor (arrow) are seen along with numerous small areas of perivascular laking (arrow with star).

pearance and distribution. An arteriogram of the specimen of a stomach containing a large leiomyosarcoma showed the same characteristics as seen in polypoid gastric carcinomas.

DISCUSSION

The definition of the location, extent and nature of internal lesions of the intestinal tract, without opening it, is surgically important. Operative arteriography promises to provide a fairly accurate way of doing this. The method, although seemingly simple and straightforward, is nonetheless somewhat time consuming. It often requires mobilization of the bowel or stomach in order to place the film cassettes in proper contact. It would be easier to have the film under the patient in a Bucky device but with the present technique this gives much poorer detail. Differences in the vascular pattern of a benign adenoma of the colon and a polypoid carcinoma are very fine, and any sacrifice in detail may lead to the wrong diagnosis.

It is difficult to draw final conclusions on the basis of studies done to date. Undoubtedly, much more work will be needed

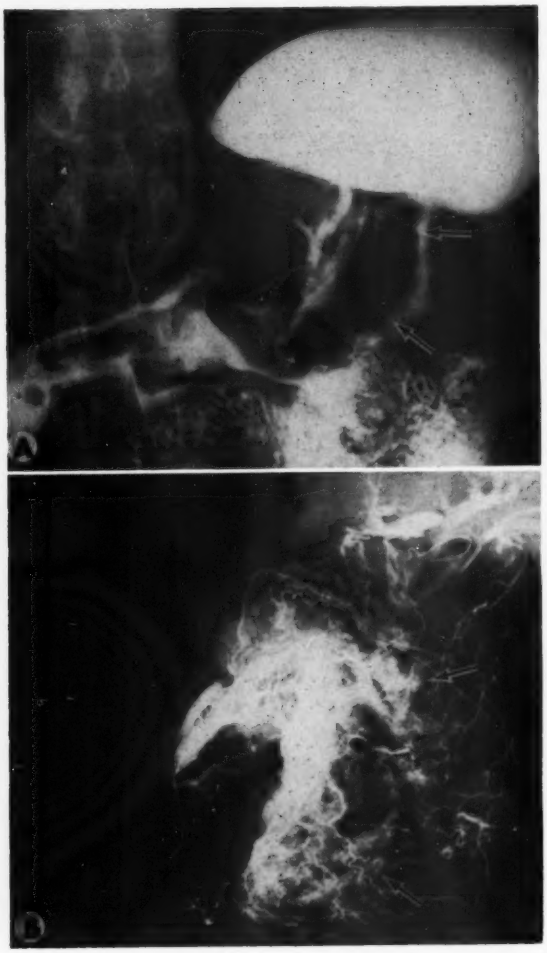

FIG. 7. Large polypoid carcinoma of the stomach. (*A*) A supine study with barium. The arrows point to the lateral margin of the mass. (*B*) An arteriogram of the dissected portion of the stomach. Within the tumor are numerous irregular vessels and large areas of opacification as well as those of perivascular density. The extent of the tumor is outlined by arrows.

before definite vascular patterns for various types of neoplasms of the stomach, small bowel and colon are established and before the validity of diagnostic criteria of this method survives repeated testing. The criteria arrived at from our material so far are given in Table II.

Besides the obvious utility of operative mesenteric arteriography for differentiation of various tumors and inflammations of the intestinal tract, it is, at times, very useful for the location of the site of massive gastrointestinal hemorrhage. We have already reported its use in this regard.[3] So far, we have used operative mesenteric arteriography only on the parts of the gastrointestinal tract that the surgeon has already decided to remove, having been worried about the toxic propensities of the contrast materials available. Evaluating the safety of this procedure in the dog, we[2] have found that methylglucamine diacetylamino-triiodobenzoate (renografin) is the safest contrast medium of those studied. Eight to ten milliliters of 40 per cent renografin in saline appear to be a safe amount to inject into a loop of bowel which is not to be resected.

SUMMARY

The method and findings of operative mesenteric arteriography are presented. Data obtained from injections of operative specimens of the colon and stomach are also referred to. Vascular patterns obtained by arteriography of malignant and benign tumors of the colon, chronic ulcerative colitis, carcinoma of the stomach and gastric ulcers are described.

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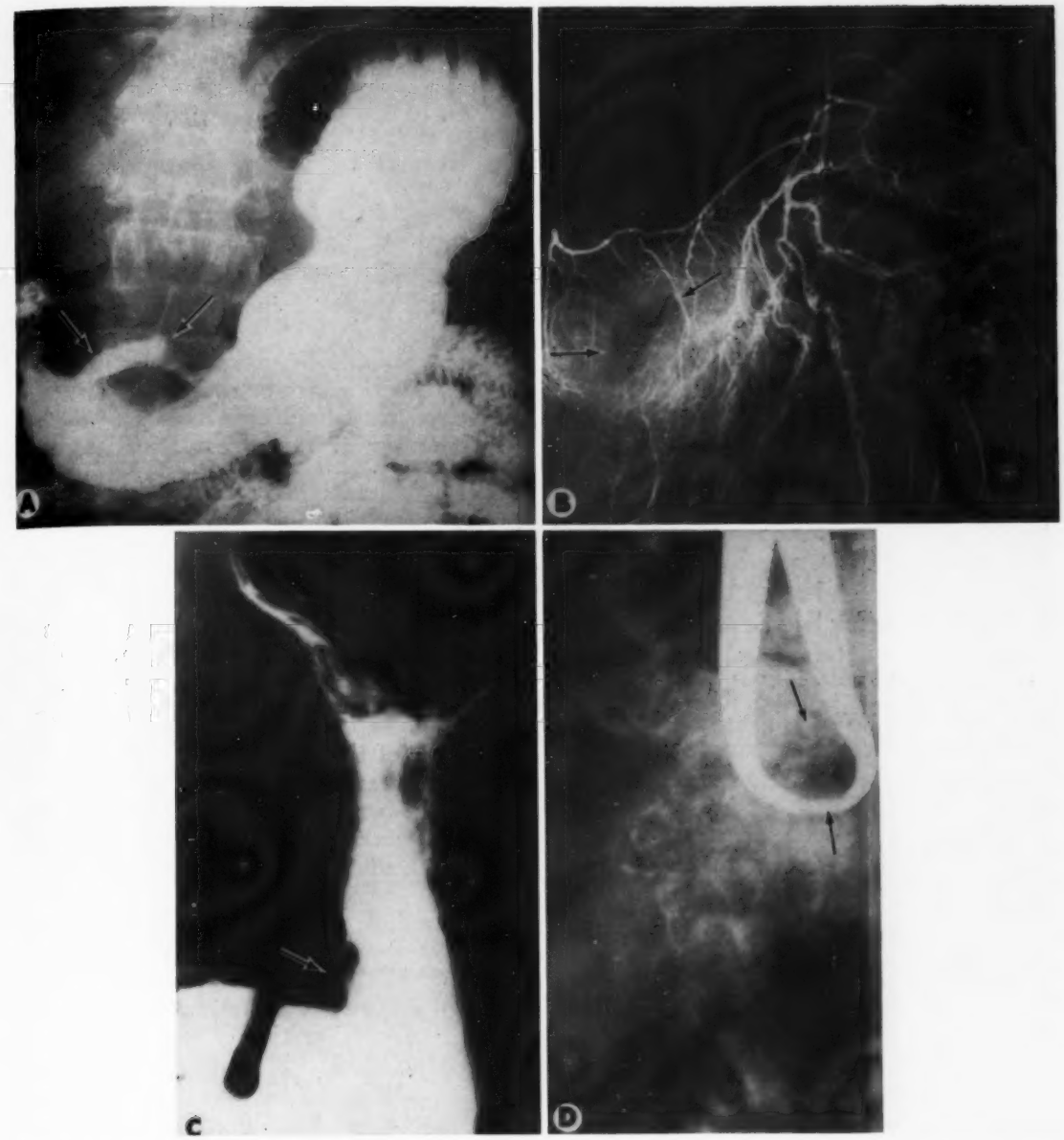

Fig. 8. (*A* and *B*) Large ulcerative carcinoma of the stomach. (*A*) A roentgenogram of the upper gastrointestinal tract with barium. (*B*) Only few vessels are seen in the area involved by the tumor. There is a strong suggestion of some irregularity in the lumen of the many attenuated vessels. (*C* and *D*) Benign peptic ulcer of the lesser curvature of the stomach. (*C*) An upright roentgenogram of the barium filled stomach with the ulcer shown in profile (arrow). (*D*) An operative arteriogram. Numerous tortuous vessels surround the ulcer which is the radiolucent area (arrows). There is no evidence of perivascular opacity. (A metallic instrument was left in the field of exposure by error.)

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URETERAL OBSTRUCTION FROM REGIONAL ENTERITIS*

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URETERAL obstruction from regional enteritis is unusual. However, familiarity with this possibility can result in a correct preoperative diagnosis of enteritis, even in the absence of dominant intestinal symptomatology. The purpose of this paper is to report 2 cases illustrating this fact.

Urologic complications as a result of intestinal disease are well known.[8] They are usually caused by colonic disease, with abscess and fistula formation as the most common complications. Less well known is the fact that ureteral obstruction can occur as a result of regional ileitis without abscess formation.[7]

Hyams *et al.*[6] reviewed the literature to 1943, including over 1,000 cases of ileitis, and describe what is apparently the first case of ureteral obstruction from this disease.

Ginzburg and Oppenheimer[4] report 13 urologic complications in 300 cases of regional enteritis, but all of these were fistulae with abscess formation. Narath[8] mentions many extrinsic inflammatory causes of ureteral obstruction but not enteritis. Most urologic and gastrointestinal roentgenologic texts[1,2,5] do not mention the association of regional enteritis and ureteral obstruction. Redish[9] in 1952 reported 6 cases of ureteral obstruction due to regional enteritis. Two of these patients, who were known to have regional enteritis, presented with right urinary tract complaints. The other 4 cases were found upon survey study of a group of 22 patients with known enteritis and no urinary symptoms.

REPORT OF CASES

CASE 1. W.O'B., a thirty-three year old white woman, was admitted to Misericordia Hospital, Philadelphia, on April 22, 1957 complaining of right lower quadrant pain. The patient had been well until one month prior to admission when she developed mild, intermittent, colicky pains in the right lower quadrant of the abdomen which were associated with frequent, non-bloody, loose bowel movements and occasional nausea and vomiting. The positive physical findings were limited to the patient's abdomen. Tenderness associated with rebound and percussion tenderness were present in the right lower quadrant. There were no organs or masses palpable. Peristalsis was normal. Slight tenderness was present in the right flank. All laboratory examinations were within normal limits.

The right lower quadrant pain and tenderness gradually subsided during the first three days of hospitalization. An intravenous urogram revealed marked dilatation of the upper two-thirds of the right ureter associated with calyceal dilatation of the right kidney (Fig. 1). Retrograde urography demonstrated similar findings in addition to a partial ureteral obstruction at the level of the first sacral vertebra. A barium enema examination showed a large, rounded, filling defect in the right transverse colon just distal to the hepatic flexure (Fig. 2).

After suitable preparation, the patient underwent exploratory laparotomy on May 4, 1957 at which time the distal 20 cm. of the ileum was found to be involved in one large inflammatory mass. The bowel was markedly erythematous, edematous and friable. This large mass lay in the right paravertebral gutter and was obviously compressing and distorting the right ureter. The mass was also adherent to the right transverse colon. The terminal 20 cm. of ileum, along with the ascending colon and the right half of the transverse colon, was resected and an end-to-end ileotransversecolostomy was accomplished. The patient withstood this operative procedure well and subsequently had an uneventful recovery. A postoperative intravenous urogram revealed a normal right upper urinary tract. The pathologic report was regional enteritis.

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Case II. N.U., a twenty-nine year old white woman, was admitted to Misericordia Hospital, Philadelphia, on September 24, 1959 complaining of right upper abdominal pain associated with nausea and vomiting. Two months prior to admission the patient, in her usual state of good health, experienced the first attack of crampy epigastric and right upper quadrant pain which radiated to the right subcostal area. An episode of diarrhea without melena occurred at this time and lasted for one week. Several similar attacks occurred during the ensuing weeks prior to admission. On the day before admission, following the ingestion of fried hamburger the patient experienced another episode of epigastric and right upper quadrant abdominal pain and vomited most of the ingested food. Chills and fever were present for two days prior to admission.

In the past the patient had undergone an appendectomy and inguinal herniorrhaphy. An undetermined type of "rectal operation" also had been performed some years previously. Upper gastrointestinal roentgenograms and a cholecystogram performed one month prior to admission at another institution were reported as normal.

Fig. 2. Case 1. Barium enema examination demonstrates a filling defect in the proximal transverse colon in the right midsacral area, the same region as the ureteral obstruction.

Fig. 1. Case 1. Intravenous urogram shows right ureteral obstruction at the level of the midsacrum.

The positive physical findings were limited to the abdomen, where the scars of previous surgery were noted. The abdomen appeared tense and somewhat rounded. Complete evaluation was difficult because of voluntary muscle guarding. There was moderate tenderness in the right upper quadrant and right periumbilical areas. There was slight tenderness in the right flank. No masses or organs were palpable.

During the first five days after admission the patient's fever ranged from 100° to 103° F. This gradually subsided spontaneously without specific treatment. Small doses of narcotics were necessary for the control of continued right upper quadrant and right periumbilical crampy pain. The tenderness persisted in these areas but was never acute.

Laboratory investigation revealed a normal hemogram and urinalysis. An upper gastrointestinal series and cholecystography showed findings within normal limits. Intravenous urography revealed a moderate distention of the calyceal system of the right kidney; the right ureter was dilated in the upper half and an area of constriction was visualized in the midureter (Fig. 3). This was thought to represent extrinsic compression. Right retrograde

FIG. 3. Case II. Preoperative intravenous urogram shows a dilated right ureter down to the upper sacral area.

urograms confirmed the findings of an extrinsic pressure defect in the ureter at the level of the body of the fourth lumbar vertebra (Fig. 4 and 5). Considerable distention of the small bowel with numerous air-fluid levels was evident on plain roentgenograms of the abdomen (Fig. 6 and 7). A diagnosis of intestinal disease, possibly enteritis, causing urinary obstruction was suggested.

At laparotomy on October 12, 1959 approximately eighteen inches of adherent, inflammed and edematous bowel was found in the right paravertebral gutter in the region of the fourth lumbar vertebra. The adjacent mesentery was thickened, edematous and contained numerous enlarged lymph nodes. This large mass of inflammed small intestine and mesentery was intimately adherent to the right ureter, which resulted in its distortion and partial obstruction. Two additional "skip areas" of involvement, each measuring about six inches, were found in the proximal portion of the ileum. The small intestine proximal to these areas was edematous and dilated but not obviously inflammed. These dilated fluid-containing loops extended from the most proximal area of involvement up to the ligament of Treitz. The involved segment of ileum was excluded from the intestinal tract and a bypass ileotransversostomy was accomplished. The patient tolerated the operative procedure well and subsequently experienced an uneventful postoperative course.

Prior to the patient's discharge, intravenous urography revealed a normal right upper urinary tract (Fig. 8). The patient has enjoyed good health in the six month period since the operation. During this time she has been completely free of abdominal complaints and has gained five pounds in weight. An initial diarrhea of six to seven bowel movements daily has gradually subsided. Currently, the patient has one to two soft formed bowel movements daily. Intravenous urography revealed a normal urinary tract six months following operation.

DISCUSSION

Urinary tract involvement in intestinal disease is well known.[8] Most of the reported complications have been abscess and fistula formation and have been due to

FIG. 4. Case II. Right retrograde urogram shows slight dilatation of the upper two-thirds of the right ureter. Note the distended small intestine in the right upper abdomen.

malignant disease and diverticulitis of the colon. Abscess and fistula formation as a result of regional enteritis are not common, occurring in about 3 per cent of large series of cases.

Ureteral obstruction as a result of regional enteritis without abscess formation is distinctly uncommon, but at least 7 cases have been reported.[4,6,9] In most of these cases the right ureter has been obstructed. In 1 case both ureters were involved. All of the previously reported cases were in men. Our 2 cases were in young women. In 3 of the reported cases, the patient presented with right urinary tract

FIG. 5. Case II. Right retrograde urogram in the oblique view showing constriction in the middle third of the ureter.

FIG. 6. Case II. Plain roentgenogram of the abdomen shows gaseous distention of the small intestine in the upper abdomen.

FIG. 7. Case II. Erect roentgenogram of the abdomen shows air fluid levels in the small intestine.

FIG. 8. Case II. Postoperative intravenous urogram shows that the right urinary tract has returned to normal. There is very little gas in the intestinal tract.

symptoms and the correct diagnosis was made because the presence of regional enteritis had previously been established. The 4 other cases reported in the literature were asymptomatic cases found by Redish[9] in a survey of 22 patients known to have regional enteritis.

In our first case, the diagnosis was not made until operation. Preoperatively, a diagnosis of neoplasm of the colon with retroperitoneal extension and ureteral obstruction was considered the most likely possibility. In our second case, the obstructed right ureter was noted at intravenous urography early in the study. During this same period, changing partial small bowel obstruction was noted on the abdominal roentgenograms. With our knowledge of the previous case two years before, the association of small intestinal obstruction and right ureteral obstruction suggested the preoperative diagnosis of regional ileitis. This was confirmed at laparotomy.

The partial ureteral obstruction in these 2 cases was due to edema associated with the terminal ileitis. The involved ileal segment in each instance was adherent to the retroperitoneal area with loose inflammatory adhesions. In Case I it was also adherent to the colon.

We believe that when a young adult is seen with partial right ureteral obstruction and partial small bowel obstruction the diagnosis of regional ileitis should be considered.

SUMMARY

Urinary tract involvement with regional enteritis is unusual. Partial ureteral obstruction in the absence of abscess formation is rare. Two cases are reported and added to those previously recorded in the literature.

We believe that, when a young adult is seen with roentgenographic evidence of partial right ureteral and small bowel obstruction, the diagnosis of regional ileitis should be considered.

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MEDULLARY SPONGE KIDNEY*

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LINDVALL,[2] in a review of the literature and a report of 35 cases, discusses many aspects of medullary sponge kidney and, in particular, its roentgenologic features. On the basis of these 35 cases, Lindvall suggests that the disease is not as rare as the scant literature on the subject and the single case reports would indicate. Until this article was published, the roentgenographic appearance of this renal lesion was not generally appreciated. This, despite the fact that the clinical and roentgenologic features of the disease had been described by Lenarduzzi[1] in 1938. The term "medullary sponge kidney" was coined by Lenarduzzi to describe the appearance of the renal lesion.

The disease morphologically is an ectasia or cyst-like dilatation of the collecting ducts of the renal pyramids and hence is a lesion of the renal medulla (Fig. 1).

Recently, we have had the opportunity of seeing a patient (Case XI) with this renal lesion who died of unrelated disease. Autopsy examination permitted pathologic confirmation of the diagnosis. A review of our records revealed another autopsied case (Case XII) with the unequivocal gross and microscopic anatomic findings of medullary cystic disease of the kidneys. A third case (Case X) with the classic roentgen features of this lesion died in 1956 of unrelated disease. A review of the pathologic material available was unsuccessful in demonstrating the specific histopathologic findings of this disease, although at the time of the autopsy mention was made of some intratubular and interstitial calcification.

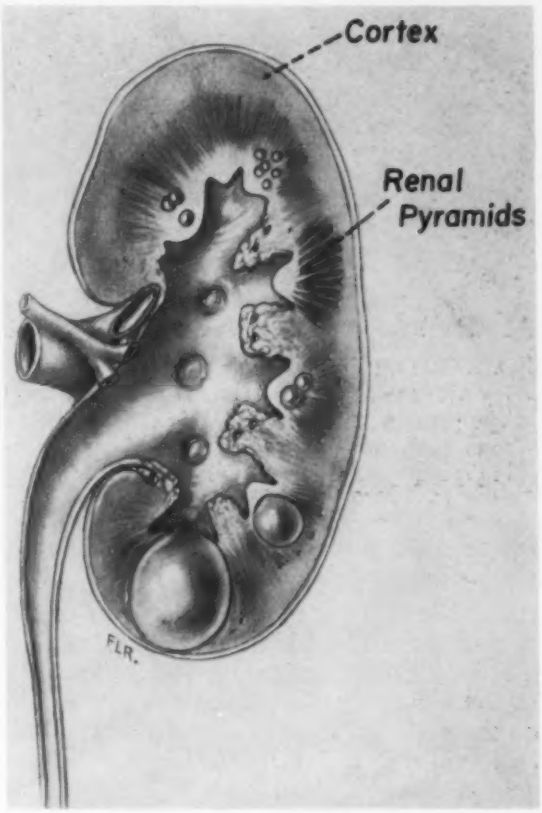

FIG. 1. Schematic drawing showing various stages of ectasia of the ducts of the renal pyramids. These range from minimal widening of ducts to formation of small cysts, then large cysts, many of which are shown connecting with calyces.

REPORT OF CASES

CASE 1. L.G. (N.Y.H. #489999), a forty-five year old Negro male, had enjoyed good health until five months prior to hospital admission when he first noticed his urine to be dark amber with occasional brown flecks. There were no other urinary tract abnormalities and review of the systems was otherwise unremarkable. Physical examination revealed no abnormalities except for an elevated blood pressure of 195/120. It was stated that he was found to have had an elevated blood pressure on examination nine years previously.

Laboratory Findings. Urinalysis showed a faint trace of albumin, 2–4 red blood cells and 6–12 white blood cells per high power field.

* From the Department of Radiology, The New York Hospital—Cornell Medical Center.
Presented at the Sixty-first Annual Meeting of the American Roentgen Ray Society, Atlantic City, New Jersey, September 27–30, 1960.

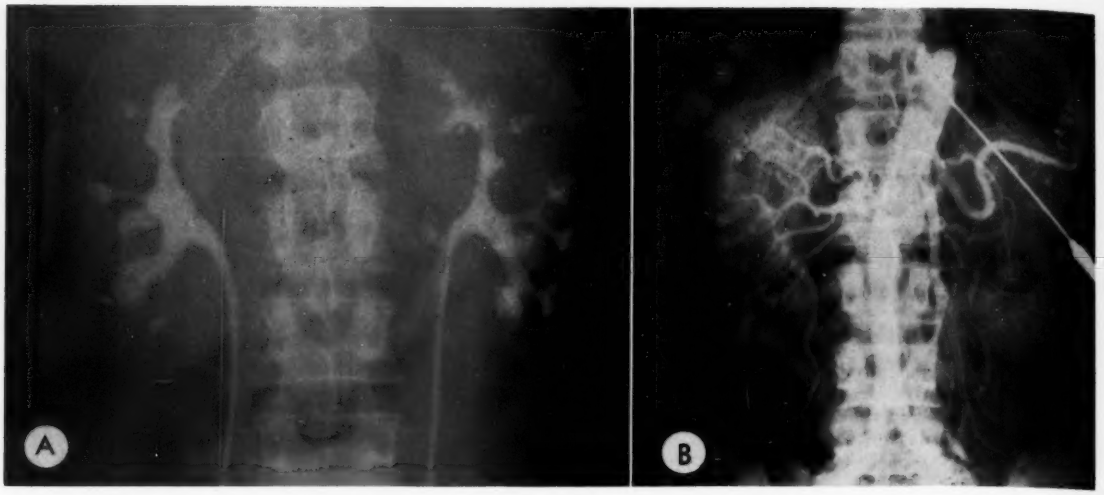

Fig. 2. Case I. (*A*) Retrograde pyelogram shows the small cysts and dilated collecting tubules communicating with the calyces in the renal pyramids. Urograms over the succeeding four years showed no change. The pyelographic image is highly characteristic of medullary sponge kidney. (*B*) Retrograde aortogram, second injection, reveals normal renal vasculature. Small discrete collections of contrast medium are seen in the cystic collecting tubules representing residua from the first injection.

Cultures were negative for acid fast bacilli. Papanicolaou stain was negative for malignant cells. Sickle cell preparations showed a 60 per cent sickling; blood chemistries were normal.

Roentgen Studies. Intravenous pyelography on October 17, 1956 revealed no opaque calculi on the preliminary scout roentgenogram. Following injection of opaque medium there was prompt bilateral excretion without any evidence of hydronephrosis. The striking finding, however, was the presence in both kidneys of minute collections of the opaque material within the collecting tubules of the renal papillae. In addition, some of the minor calyces were surrounded by an amorphous blush of the contrast material. A retrograde pyelogram on November 9, 1956 demonstrated the tubular ectasia and cysts to excellent advantage (Fig. 2*A*). The true nature of the lesion was not understood at this time and the roentgenologic diagnosis was acute necrotizing papillitis, although the patient's clinical course and subsequent roentgen examinations were not compatible with this diagnosis. A direct translumbar aortogram (Fig. 2*B*) demonstrated both renal arteries to be grossly normal.

Follow-up studies on this patient in 1957, 1958 and 1959 showed no essential change in the appearance of the kidneys on the interval urograms.

The patient has not developed demonstrable calculi. There have been no further episodes of hematuria. The blood pressure remains elevated but the patient has enjoyed good health.

Case II. R.T. (N.Y.H. #821215) a twenty-nine year old male, was seen by his doctor because of the recent occurrence of occipital headaches and a sense of tension. The headaches were of short duration and required no medication. The patient, otherwise, enjoyed good health. There were no urinary complaints. Physical examination revealed a normal healthy appearing adult whose examination disclosed no abnormalities except for a slightly elevated blood pressure of 136/98. Urinalysis and other routine laboratory studies were normal.

Roentgen Findings. An intravenous pyelogram demonstrated a large left kidney, no opaque calculi, prompt excretion and a startling collection of contrast agent in varying sized cyst-like cavities in the renal papillae of both kidneys (Fig. 3*A*). Many of the papillae were grossly enlarged, particularly throughout the left kidney. A retrograde study showed to good advantage filling of the various cysts (Fig. 3*B*). A delayed roentgenogram (Fig. 3*C*) revealed retention of the opaque medium in the cysts after emptying of the calyces. Nephrotomograms (Fig. 3, *D* and *E*) demonstrated in dramatic fashion the hyperplastic left kidney, the cysts and the large papillae.

Case III. J.C. (N.Y.H. #654606), a twenty-seven year old white female, was seen in our Out Patient Department for symptoms of acute cystitis, a condition for which she had been treated ten years previously. Physical examination revealed no significant abnormalities.

Laboratory Findings. Urinalysis showed a trace of albumin and an abundance of white blood cells. The other routine laboratory studies were normal

The patient responded quickly to appropriare therapy for her cystitis.

Roentgen Findings. During her workup, an intravenous pyelogram showed normally functioning, nonhydronephrotic kidneys. Both kidneys exhibited many striate densities in relation to the fornices of the minor calyces. In addition to the radiating densities, there were small cyst-like collections of the opaque material in a few papillae (Fig. 4, *A* and *B*). The examination was done without the use of ureteral compression.

Case IV. L.C. (N.Y.H. #761842), a forty-seven year old Negro male, entered the hospital because of hematuria and dysuria of two months' duration. Physical examination revealed no significant findings.

Cystoscopy revealed a tumor on the left an-

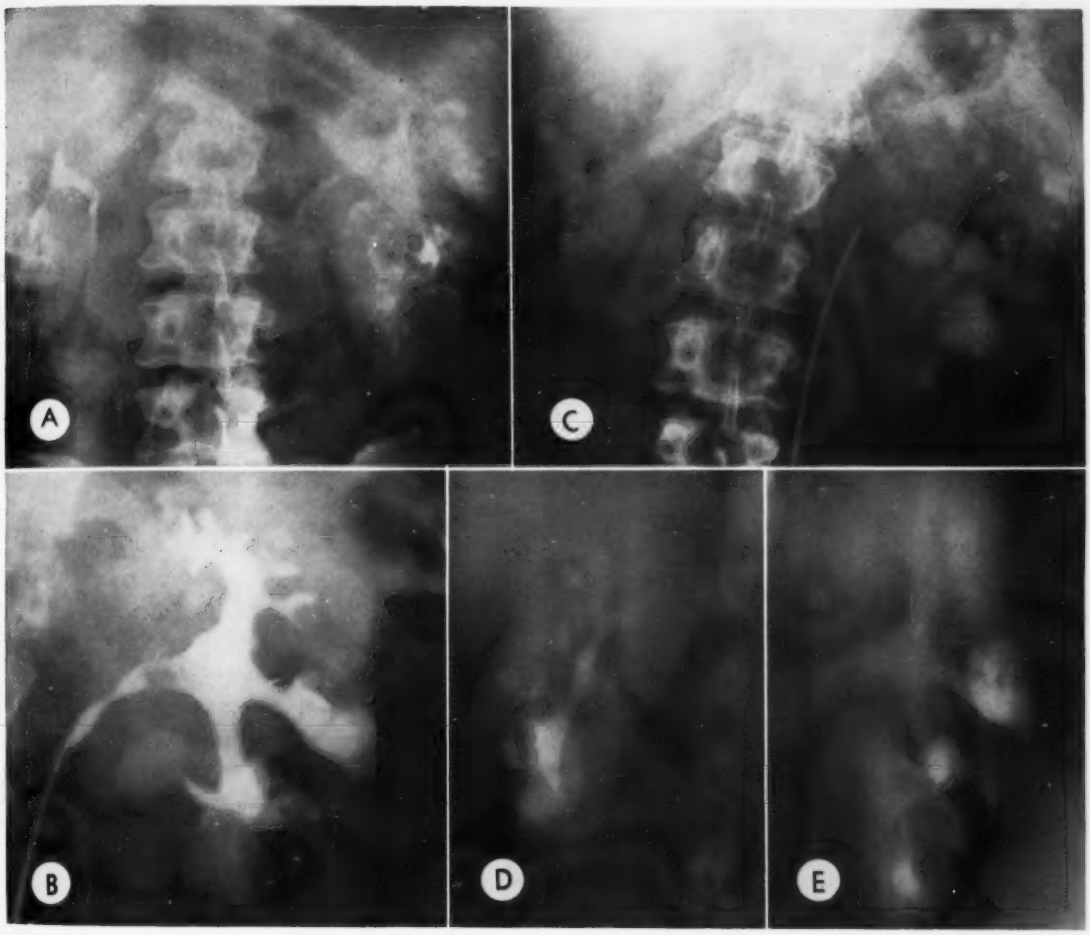

Fig. 3. Case II. (*A*) Intravenous pyelogram demonstrates bizarre collections of contrast medium in the ducts of the large hyperplastic renal pyramids. Beginning filling of large medullary cysts is noted. (*B*) Retrograde study shows the large left kidney and the presence of many large and small cysts of the renal pyramids connecting with the calyces. (*C*) Delayed roentgenogram after the retrograde study shows the retention of the contrast medium in the medullary cysts after most of it has been excreted from the collecting system. (*D* and *E*) Nephrotomograms of the right and left kidneys, with marked hyperplasia of the left. Multiple medullary cysts are seen in both kidneys. Large papillae (arrow) are also demonstrated.

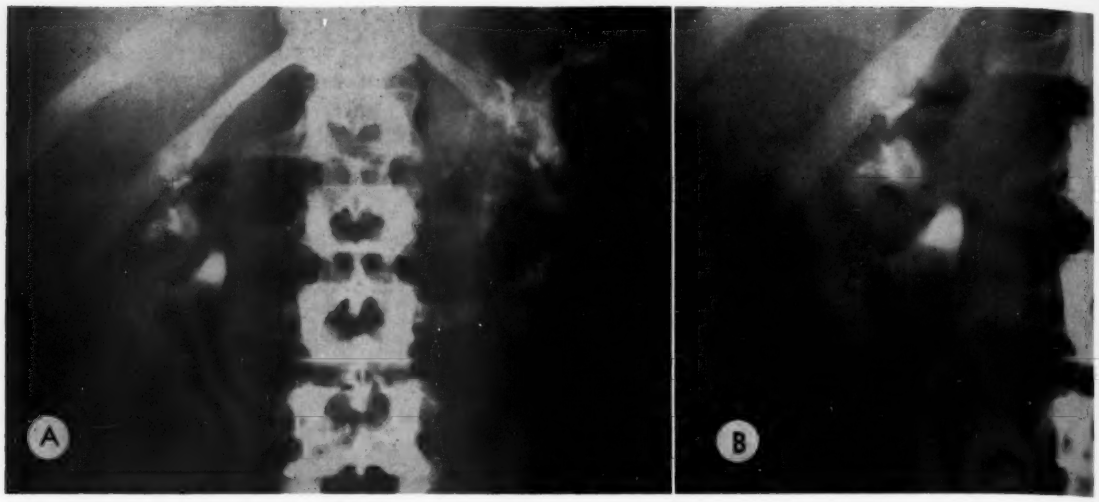

Fig. 4. Case III. (*A*) Intravenous pyelogram shows contrast medium in dilated collecting ducts of both kidneys, most pronounced on the right. (*B*) The spray-like appearance of the contrast medium in the collecting tubules of the right kidney is demonstrated.

terior wall of the bladder. There was a clear efflux from both ureters. Biopsy of the bladder tumor revealed carcinoma.

Laboratory Findings. Urinalysis showed gross and microscopic hematuria, a few white blood cells and traces of albumin. Repeated cultures and smears were negative for acid fast bacilli. Urine was also negative for heavy metals.

Roentgen Findings. An intravenous pyelogram revealed a few small punctate calcifications in the lower pole of the left kidney. The postinjection roentgenograms demonstrated prompt bilateral excretion. Surrounding each of the minor calyces were small collections of contrast agent having both a striate and nodular appearance. These were located in the papillae and at the time of this examination suggested necrotizing papillitis. There were no other symptoms to support this diagnosis and subsequent roentgen studies showed no progression or change in the appearance of the kidneys. The retrospective appearance is quite typical of what Lindvall has described as medullary sponge kidney (Fig. 5, *A* and *B*).

The patient, despite aggressive surgery and radiation therapy, expired from his disease approximately five months after being first seen. Autopsy permission was not granted.

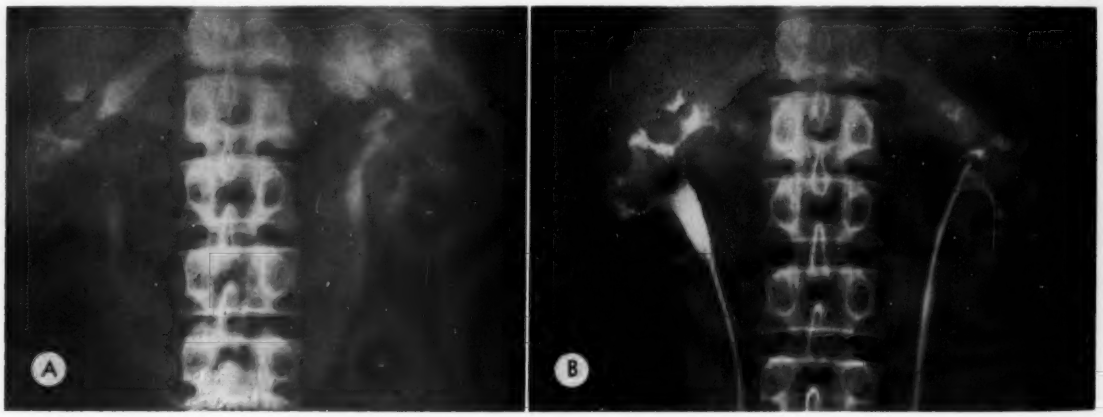

Fig. 5. Case IV. (*A*) Intravenous pyelogram shows the minor calyces to appear fuzzy and indistinct because of collections of the contrast agent in the collecting tubules of the renal papillae. This appearance is very similar to the early stage of necrotizing papillitis. (*B*) Retrograde study shows filling of dilated, ectatic, cystic collecting tubules of the renal pyramids.

Case v. A.F. (N.Y.H. #753366), a thirty-seven year old nurse, had intravenous pyelography because of slight elevation of diastolic pressure noted on a pre-employment examination. Her blood pressure was 130/90; physical examination showed no significant findings.

Laboratory Findings. Urinalysis and other routine laboratory studies were normal.

Roentgen Findings. An intravenous pyelogram revealed a small ring-like calcified density over the upper pole of the right kidney. The postinjection studies localized this in one of the minor calyces of the upper pole. In addition, striate collections of the opaque material were seen in the papillae of the calyces in the lower pole of this kidney, representing contrast agent in the collecting ducts of the papillae. The left kidney demonstrated a small cyst-like collection of the opaque medium adjacent to one of the minor calyces of the middle calyceal group. A few of the papillae of the right kidney were somewhat enlarged. Ureteral pressure was not used. The findings of calcification, visualization of collecting ducts, somewhat enlarged papillae and a cyst-like collection in the left kidney are sufficient to warrant the roentgenologic diagnosis of medullary sponge kidney (Fig. 6).

Case vi. B.R. (N. Y. H. #745315), a forty-four year old female, was admitted to the hospital for surgery of a carcinoma of the breast. The pertinent past history revealed that three–four years prior to admittance she had dysuria with several episodes of hematuria and concurrent suprapubic discomfort before voiding. Cystoscopy at that time disclosed "polyps." At the time of her present admittance she had stress incontinence, frequency and incomplete voiding.

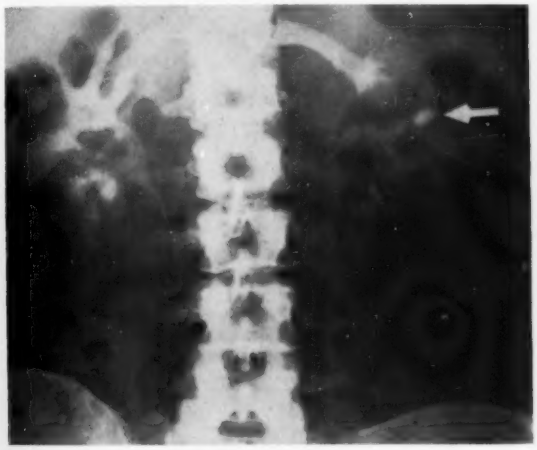

Fig. 6. Case v. Intravenous pyelogram demonstrates tubular ectasia producing spray-like, striate densities in the renal pyramids. The minor calyces are somewhat exaggerated, reflecting slight enlargement of the renal pyramids. There is a clearly defined medullary cyst in the left kidney (arrow).

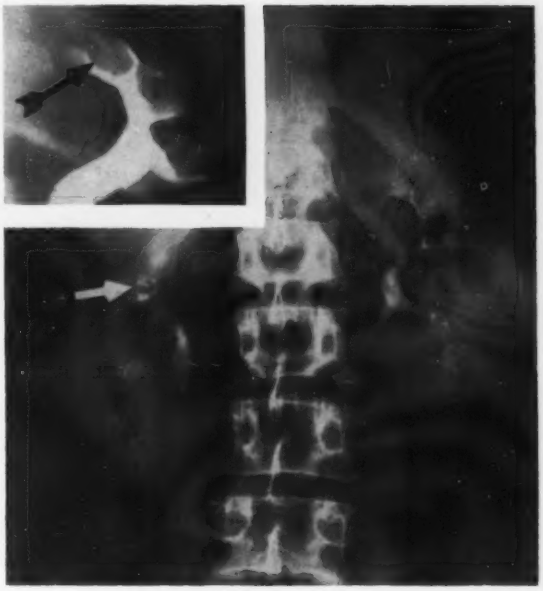

Fig. 7. Case vi. Intravenous pyelogram demonstrates opaque medium in the dilated collecting tubules, producing radiating linear densities extending from the minor calyces. Some of the minor calyces, due to enlargement of the papillae and nonfilling, have what Lindvall has referred to as a "blistered" look (white arrow). Inset demonstrates the large deeply cupped minor calyces due to enlargement of papillae.

Laboratory Findings. Urinalysis was negative; serum calcium and phosphorus were normal.

Roentgen Findings. An intravenous pyelogram showed prompt function of nonhydronephrotic kidneys. The contrast medium was seen in the collecting tubules of the papillae of both kidneys. Some of the papillae had a clear ring-like appearance that Lindvall has referred to as a "blistered look." A retrograde study showed no reflux of the contrast material into the collecting tubules but did show the papillae to be somewhat large. This was demonstrated by the rather deeply cupped appearance of the minor calyces (Fig. 7).

Case vii. I.A. (N. Y. H. #816719), a twenty-nine year old Negro woman, was found to have a slight elevation of blood pressure during

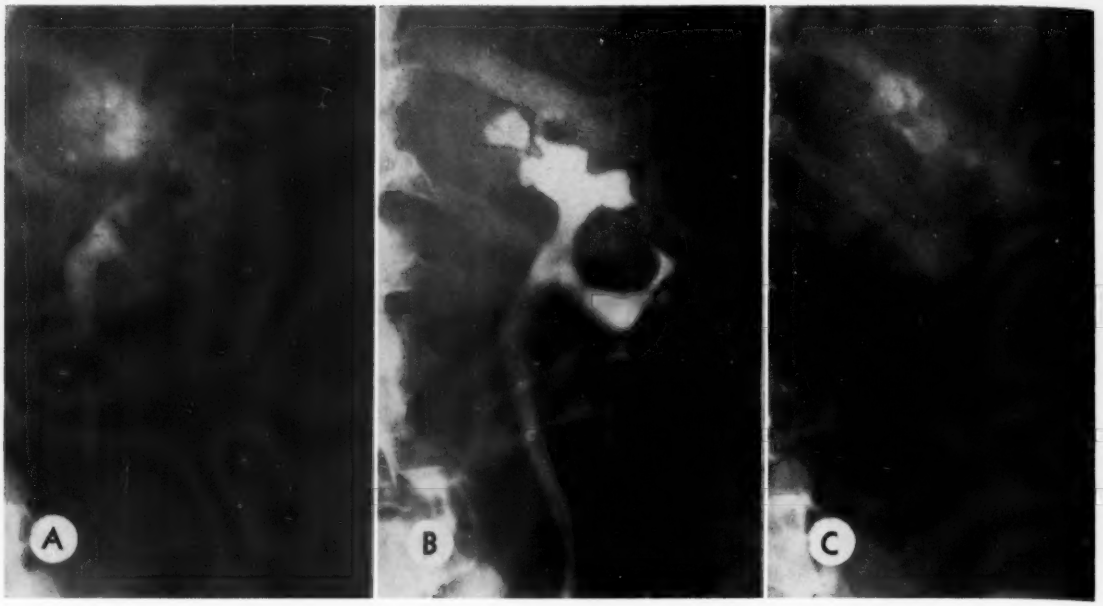

Fig. 8. Case vii. (*A*) Intravenous pyelogram shows the contrast medium in the large cysts of the renal pyramids of the upper pole of the left kidney. Many small cysts connecting with calyces are seen. (*B*) Retrograde study of the left kidney shows filling of only some of the cysts, one of which looks like a calycine diverticulum. (*C*) Delayed roentgenogram shows retention of the opaque medium in the cysts and emptying of the calyces and renal pelvis.

pregnancy. During the course of a workup to evaluate the blood pressure, roentgenograms were made of the upper urinary tracts.

Laboratory Findings. Urinalysis was negative for red blood cells, white blood cells, albumin or casts. Cultures were negative for acid fast bacilli. Blood chemistries were normal.

Roentgen Findings. An intravenous pyelogram revealed deposits of contrast medium in many of the papillae of the left kidney, arranged in radiating strand-like densities and smudgy amorphous collections surrounding the minor calyces. Retrograde pyelograms showed many small cysts in the pyramids of the left kidney (Fig. 8, *A*, *B* and *C*). An examination six months later demonstrated the same pyelographic findings now considered to be characteristic for medullary sponge kidney.

Case viii. A.M. (N.Y.H. #797519), a twenty-three year old white female, is a well documented case of lupus erythematosus. During the course of several hospital admissions she was found at roentgenography to have bilateral nephrocalcinosis.

Laboratory Findings. Urinalysis showed occasional red blood cells, 8–12 white blood cells, and a few granular casts. The sedimentation rate was 9 mm. Blood chemistries showed the blood urea nitrogen, sugar, urea clearance, phenolsulfonphthalein and Remond-Smith test to be normal.

Roentgen Findings. A review of the intravenous pyelograms revealed normal bilateral function and no hydronephrosis. Upon close inspection, however, in addition to calcific deposits in the renal pyramids, dilatation of the collecting tubules was noted (Fig. 9).

Case ix. P.F. (N.Y.H. #705577), a fifty-four year old white male, had a long history of renal disease marked by periodic exacerbations of hematuria and pain. The patient has had thirteen hospital admissions and has been treated for gouty nephropathy, chronic pyelonephritis and hypertension.

Intravenous pyelograms over this long period of observation have shown evidence of chronic pyelonephritis. In addition to these changes, there were seen small discrete cyst-like collections of the contrast medium as well as some smudgy collections in somewhat enlarged papillae (Fig. 10). Initially, it was thought that these changes represented necrotizing papillitis or possibly renal tuberculosis. Neither diagnosis, however, was supported by the clinical

course or laboratory findings. Cultures have been repeatedly negative for acid fast bacilli. It is now felt that the urographic findings are consistent with renal papillary necrosis as well as medullary sponge kidney.

Case x. M.S. (N.Y.H. #334337), a seventy-three year old female, had Laennec's cirrhosis and bleeding esophageal varices. A splenoportal venogram demonstrated obstruction of the portal vein and a splenorenal shunt was performed.

She did poorly postoperatively and died seven days after the operation. During her hospital workup, intravenous and retrograde pyelograms were made. The retrograde pyelogram demonstrated the presence of many small cyst-like cavities in the papillae of the renal pyramids. In addition contrast medium was seen in the dilated collecting tubules of the renal pyramids. The roentgen appearance was typical for that of medullary sponge kidney (Fig. 11).

Autopsy revealed carcinoma of the liver with invasion of the portal vein. There was metastatic tumor in the lungs and esophageal and gastric varices. The kidneys showed a number of infarcts. The areas of the cortex showed extensive atrophy of the tubules, fibrosis of glomeruli, intimal thickening of the small arteries and interstitial infiltration with lymphocytes. In the pyramids there was intratubular and interstitial calcification.

Fig. 9. Case viii. Intravenous pyelogram shows nephrocalcinosis with small calcific deposits in the collecting ducts of the pyramids. The opaque medium is seen in the dilated, ectatic ducts.

Fig. 10. Case ix. Pyelogram shows the many small communicating collections of contrast medium in the medullary portion of the kidney along with the somewhat enlarged pyramids. These findings are highly suggestive of sponge kidney and renal papillary necrosis.

Case xi. H.N. (N.Y.H. #836571), a fifty-six year old male, was admitted to the hospital because of severe hypertension which had been first noted five years prior to admission. Two months prior to admission he noted onset of excessive fatigability, frequency and nocturia, burning, urgency and ankle edema. Examination revealed an acutely ill white male with twitching in all extremities and profuse diaphoresis. An eye examination showed early papilledema, old and new exudates, flame shaped and round hemorrhages and extreme spasm of the retinal arterioles. The neck veins were distended, the heart was enlarged to the left and there was a blowing apical systolic

Fig. 11. Case x. Retrograde pyelogram demonstrates multiple small cysts in the pyramids, presenting the appearance of diverticula.

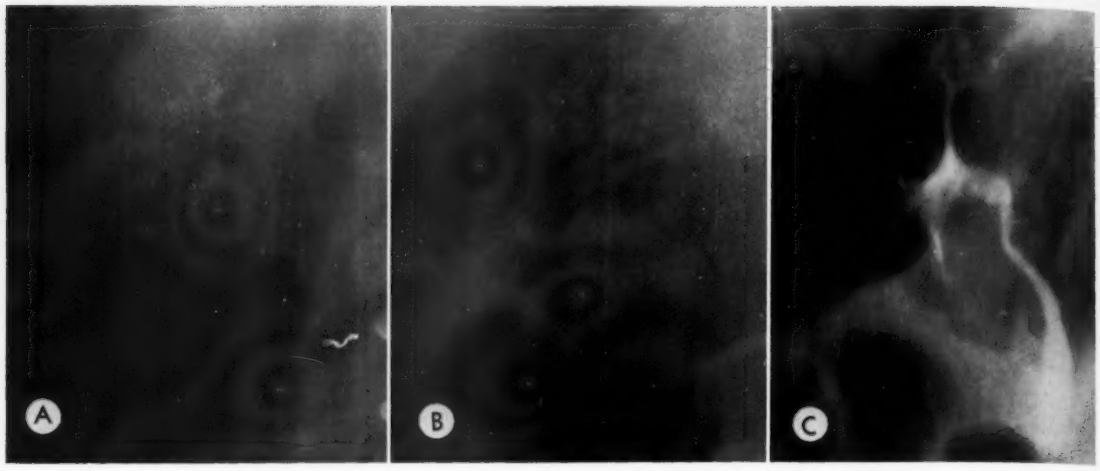

FIG. 12. Case XI. (*A*) Roentgenogram shows multiple small clusters of calcified deposits. The size, arrangement and grouping are highly suggestive of cystic lesions of the renal pyramids. (*B* and *C*) Intravenous pyelograms reveal dilated ducts and cysts of the renal pyramids of the upper pole of the kidney.

murmur. The liver was enlarged downward 4 finger breadths. His blood pressure was 200/140.

Laboratory Findings. Urinalysis showed 4 plus albumin, 10–20 red blood cells, 15–20 white blood cells per high power field and many casts. Cultures were negative for acid fast bacilli. The complete blood cell count was hematocrit 30 and white blood cells 16,600. The blood urea nitrogen, on admission and subsequently, fluctuated between 126 and 24. The electrocardiogram showed auricular fibrillation and evidence of coronary artery disease. Blood chemistries revealed evidence of hypokalemic alkalosis. The patient responded poorly to therapy for his malignant hypertension and renal failure and expired two months following his hospital admission.

Roentgen Findings. Roentgenograms of the upper urinary tracts revealed multiple small discrete calculi in the right kidney (Fig. 12*A*). Intravenous pyelograms revealed ectasia and small cyst formation of the ducts of the renal papillae in the upper pole of the right kidney. The calculi were located in the ectatic, dilated ducts (Fig. 12, *B* and *C*).

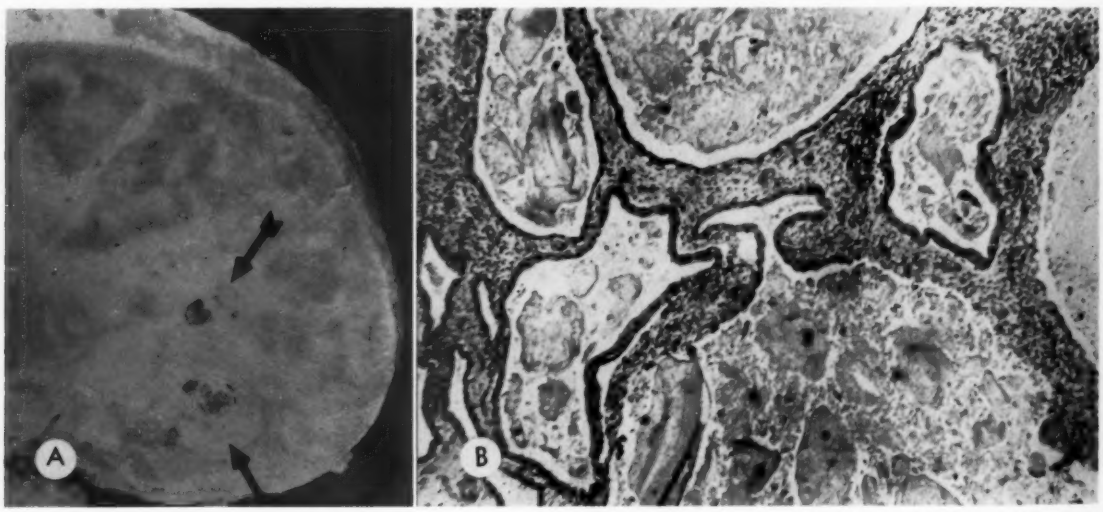

FIG. 13. Case XI. (*A*) Gross autopsy specimen shows the presence of many cysts in the pyramids of the upper pole of the right kidney (arrows). (*B*) Microscopic section shows dilated, ectatic ducts lined with low cuboidal epithelium.

At autopsy, the pertinent findings to this report were those confined to the kidneys. These revealed arterial and arteriolar nephrosclerosis; both kidneys weighed 290 gm.; finely granular cortical surfaces and uniformly thinned cortices were found. There was marked narrowing of the lumina and thickening of the walls of the arteries and arterioles due to fibrous tissue, hypertrophy of the muscularis and proliferation of the cells in the intima. The cortex of the right kidney contained thin walled cysts filled with clear fluid and measuring up to 3.5 cm. in diameter, and similar cysts, measuring up to 0.5 cm. were situated in the medulla (Fig. 13*A*). The three superior calyces of the right kidney and corresponding papillae contained brown, friable calculi. Microscopic examination demonstrated marked cystic dilatation of the medullary tubules. The cysts were lined by a single layer of low cuboidal epithelium (Fig. 13*B*).

Case xii. M.L. (N.Y.H. #339240), an eight year old girl, was admitted to The New York Hospital for the first time on September 12, 1942 because of intractable anemia. Between that date and the day of death, May 22, 1943, there were six hospital admissions. Death occurred from renal insufficiency and the anemia was directly related to the renal lesion.

At autopsy, gross examination revealed innumerable cysts sharply limited to the renal pyramids. The majority of the cysts were from 1–5 mm. in diameter. The cysts were thin walled and contained clear, colorless, odorless fluid. Microscopic examination of the renal pyramids revealed numerous thin-walled cysts among the loops of Henle and the collecting tubules, many of which were not visible grossly. There was no inflammation near the cysts. The cysts were lined by a layer of low cuboidal cells which closely resembled the cells of the collecting tubules (Fig. 14).

(This case has been reported previously.[4])

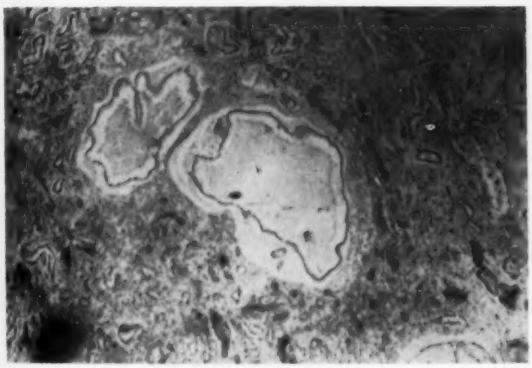

Fig. 14. Case xii. Microscopic section shows dilated, cystic, collecting tubules lined by a single layer of low cuboidal epithelium.

Discussion

The etiology of medullary sponge kidney is not completely understood. In all probability it represents a congenital defect in development of the collecting tubules in the renal medulla. The dilatation of the ducts of the renal pyramids and papillae may vary from slight widening to the formation of large cyst-like cavities. The extent of the disease also varies from involvement of one or several papillae of one kidney to diffuse bilateral involvement. The affected papillae may enlarge and result in general enlargement of the kidney.

Minimal enlargement of the collecting ducts will, on excretory urograms, be seen as spray-like densities radiating from a minor calyx (Fig. 4*B*). The appearance is similar to, but somewhat more pronounced than, that of simple stasis within the ducts. Differentiation in such cases may be possible only if there is enlargement of the involved renal pyramids. Visualization of the renal papilla as a physiologic occurrence is always a transient phenomenon, whereas visualization of the ectatic collecting ducts of medullary sponge kidney is constant and reproducible. Extreme dilatation of the ducts results in the formation of cyst-like cavities, usually connecting with the minor calyces. The roentgenographic appearance of the advanced form of the disease produces a startling bizzare picture of ectatic tubules, multiple cysts, large papillae and occasionally large kidneys (Fig. 3, *A–E*). The correct diagnosis in such instances can be made by the specific and characteristic pattern seen on the pyelogram.

The cysts are best demonstrated by excretory urography where they fill with the contrast medium ahead of the pyelocalyc-

eal system and retain the contrast agent after the calyceal system has been emptied.

Calculi, usually small and discrete, are frequently seen in the cyst-like cavities. They were present in all but 2 of Lindvall's cases and in 5 of our cases. The presence of calculi tends to provoke such complications as obstruction, hematuria and infection.

The disease is said to be confined to the medulla. In one of our autopsied cases, several cysts were also seen in the cortex and probably represented simple retention cysts.

Reilly and Neuhauser[3] recently reported 11 cases of renal tubular ectasia in infants and children with a high association of cystic disease of the liver and pancreas. The morphologic character of the renal lesions in our cases is similar to that reported by these authors, but in none of our cases was there anatomic or clinical evidence of associated cystic disease of the liver or pancreas.

The major roentgenologic considerations in the differential diagnosis are renal tuberculosis, hyperparathyroidism, necrotizing papillitis and calyceal diverticula. None of these diseases should provide serious difficulty. Necrotizing papillitis in our experience has been a fatal disease. Extensive necrosis of the renal papillae presents large ragged excavations on pyelograms. It is also usually associated with some degree of obstructive uropathy or diabetes. Nephrocalcinosis associated with hyperparathyroidism may offer some difficulty but the other stigmata of the disease are usually sufficient to differentiate it from medullary sponge kidney. The ragged cavitary defects of renal tuberculosis are not limited to the papillae; also other evidence of the disease is usually adequate to differentiate it from sponge kidney.

SUMMARY

Twelve cases possessing the roentgenographic and/or pathologic criteria of medullary sponge kidney have been presented. The anatomic and roentgenologic features of the disease have been discussed and illustrated.

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THE ROENTGEN MANIFESTATIONS OF BILATERAL RENAL CORTICAL NECROSIS*

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BILATERAL renal cortical necrosis as a cause of acute renal failure was first recognized by Juhel-Renoy in 1886.[20] By definition, this is a condition characterized by bilateral symmetric ischemic necrosis involving the cortical portions of the kidneys, including the columns of Bertini, with relative sparing of both the medulla and a rim of subcapsular cortical tissue.[13] In the past, little attention has been directed to the roentgen aspects of this disorder since its rapidly fatal course was unassociated with specific roentgenographic features.

In recent years patients with bilateral renal cortical necrosis have survived for longer periods of time due to a better understanding of this disorder and to the availability of improved supportive measures in the treatment of acute renal failure. In some instances this prolonged survival has afforded sufficient time for the mineralization of necrotic cortical tissue, an alteration detectable by roentgen means.

Although Wells, Margolin and Gall[20] state that less than 160 cases of renal cortical necrosis have been reported through 1959, undoubtedly many more cases have gone unrecognized or unreported. This figure does not include the 21 cases described by these authors. A review of the literature has disclosed 4 cases which exhibited antemortem roentgenographic findings characteristic of renal cortical necrosis.[2,6,11,15] In only one article was specific attention called to the roentgen features.[15] The present paper deals with a case of bilateral renal cortical necrosis in which, following survival of the acute episode, cortical calcification as well as shrinkage of the kidneys developed.

REPORT OF A CASE

A twenty-four year old white female was transferred from another hospital for management of acute renal insufficiency. She had been in her sixth month of pregnancy when nausea, vomiting, and abdominal cramps appeared. Her blood pressure at that time was 160/100, and she was considered to be in a pre-eclamptic state. Severe vaginal bleeding promptly followed and a stillborn fetus was delivered by emergency low cervical cesarean section. The clinical impression of placenta previa was substantiated. Considerable blood loss occurred prior to and during the operation, although no blood pressure reading below 120/80 was recorded. Four units of blood, properly cross-matched, were administered without reaction during the operation and within forty-eight hours of its termination. As a result of these transfusions, the hemoglobin rose from 4 to 10.5 gm./100 ml.

Oliguria developed immediately following the operation. Over the next five days the urine output varied between 25 and 50 cc. per day. The blood urea nitrogen rose to 135 mg./100 ml. and remained between 100 and 200 mg./100 ml. for the next thirty-five days. On the fourth postpartum day, at the time of transfer to the Cincinnati General Hospital, pulmonary congestion, peripheral edema, and abdominal distention were present. Over the next three days the serum potassium rose to 7.9 mEq./liter, and there was electrocardiographic evidence of hyperkalemia. Following hemodialysis with an artificial kidney there was considerable improvement in the blood chemistry values.

Bilateral flank pain, which appeared early in the course of her disease, subsided and the urine, which had been grossly bloody, cleared. The urinary albumin ranged from 2 to 4 plus. Antibiotic agents were administered for pyuria and bacilluria which developed as a result of an indwelling catheter which had previously been inserted.

Three days after hemodialysis acute pulmonary edema developed and her clinical condition deteriorated. Hemodialysis was repeated, and again there was clinical improvement. A

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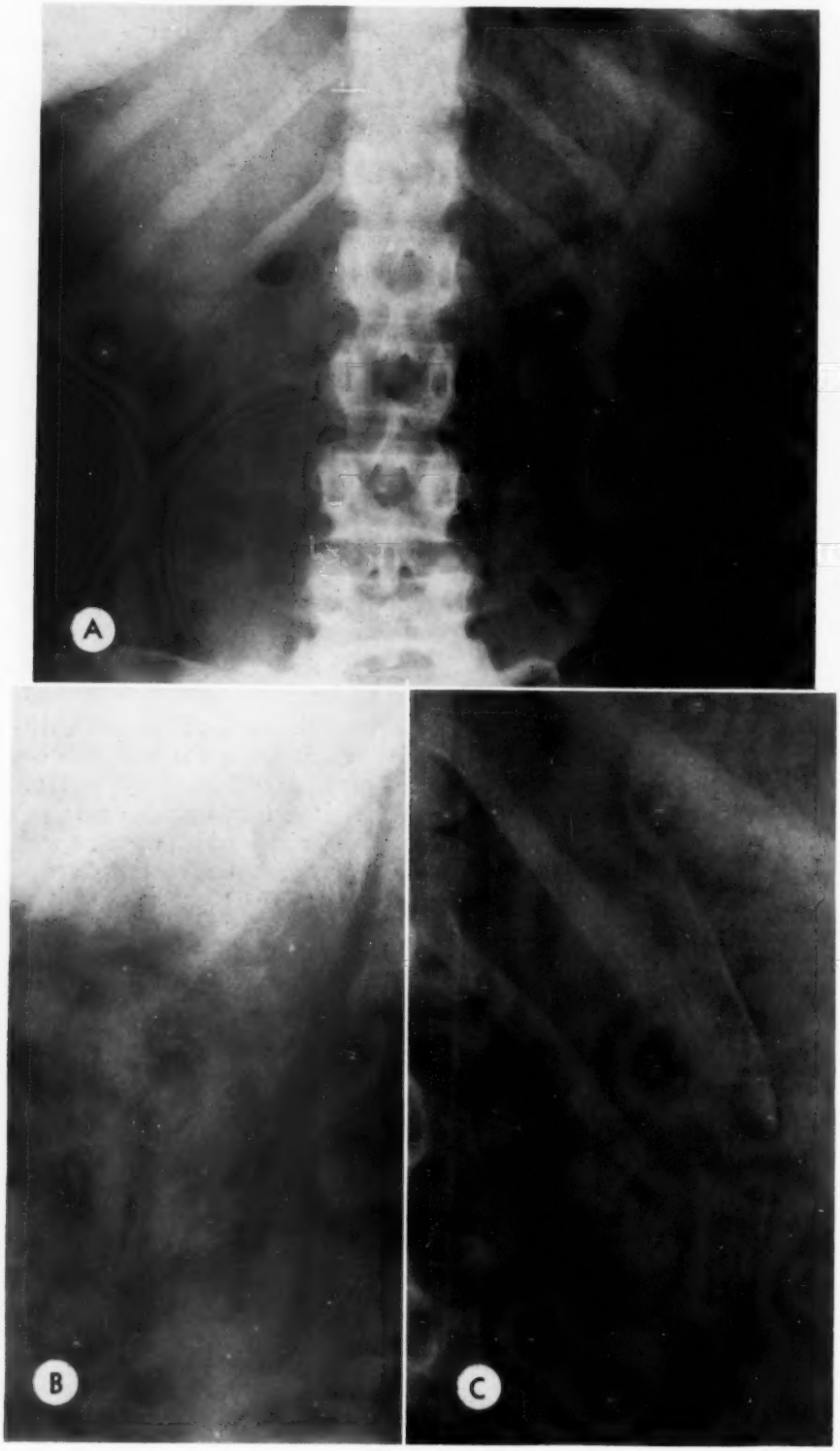

Fig. 1. (*A*) Plain roentgenogram of the abdomen taken on the fifty-seventh day of illness. A narrow rim of renal cortical calcification is seen bilaterally. The obvious splenomegaly is an incidental finding. (*B* and *C*) Oblique roentgenograms of each kidney demonstrate the cortical calcification to better advantage.

few days later pulmonary edema recurred. This was accompanied by hemorrhage from the vagina and from the site of a tracheostomy. Dehiscence of the abdominal incision occurred and was closed with wire sutures. Loud pleural and pericardial friction rubs were audible and there was cardiac arrhythmia. The mental confusion and delirium which followed the first hemodialysis did not improve.

Three weeks after the first dialysis the urinary output, which had ranged between 25 and 200 cc. per day, rose to normal levels while the blood urea nitrogen remained at about 200 mg./100 ml.; three weeks later it had fallen to 84 mg./100 ml. Anemia and splenomegaly (the latter was first noted several days following cesarean section) persisted. The anemia was attributed to chronic blood loss. Hypersplenism was not felt to be present.

On the fifty-seventh day following delivery, abdominal roentgenograms were made preliminary to a renal biopsy (Fig. 1, *A*, *B* and *C*). These revealed moderately extensive renal cortical calcifications. These were more apparent on laminagrams (Fig. 2, *A* and *B*). It was also apparent that both kidneys had decreased considerably in size when compared with roentgenograms two months earlier. Over the ensuing nineteen months the albuminuria and mild hypertension have persisted. Several bouts of urinary tract infection have been treated successfully. The blood urea nitrogen and creatinine have fallen to normal levels although low urea and creatinine clearances have been obtained repeatedly. During this period there has been a slight increase in the extent of cortical calcification and a further slight reduction in the size of the left kidney. A recent intravenous pyelogram (Fig. 3, *A* and *B*) demonstrated prompt excretion with moderate concentration of the contrast material. Normal pelvicalyceal systems were observed bilaterally.

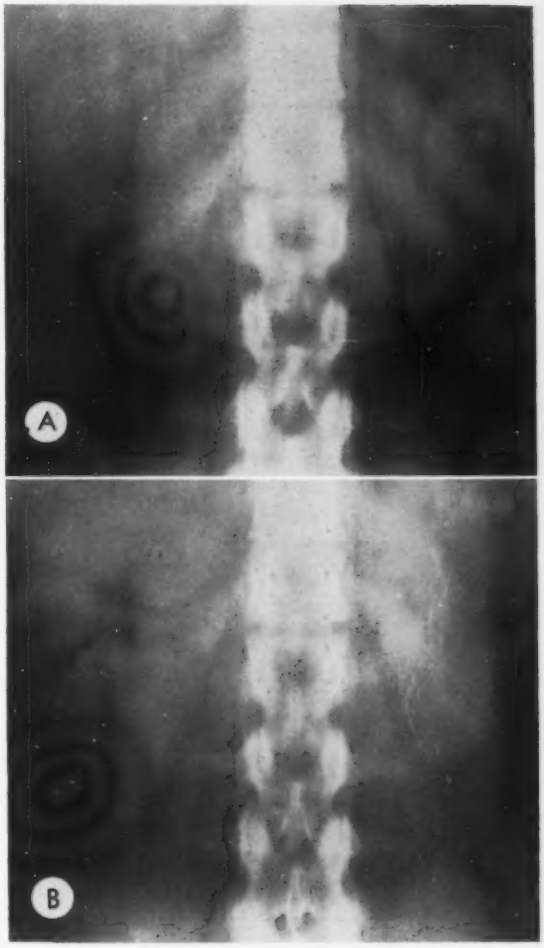

Fig. 2. (*A*) Laminagram demonstrates the calcification well and shows its cortical distribution. (*B*) Laminagram seventeen months later. There is a slight increase in the amount of calcification and a further slight decrease in the left renal size.

Discussion

Bilateral renal cortical necrosis has long been recognized as a cause of acute renal failure. A great variety of apparently unrelated conditions have been observed to antedate the abrupt onset of this disorder. It has occurred in association with severe burns, multiple fractures, internal hemorrhage, major surgical procedures, severe infections, malignant neoplasms, transfusion of incompatible blood, the administration of a variety of nephrotoxins, favism, diarrhea with dehydration, influenza, perforated peptic ulcer, peritonitis, and periarteritis nodosa. The occurrence in pregnancy (usually in association with abruptio placentae)[1,13] has been frequently emphasized in the literature. Duff and More[7] collected 71 examples of cortical necrosis through 1941 and noted that 67 per cent were associated with pregnancy. Wahle and Muirhead[19] also reviewed the literature on this subject through 1953 and observed that 57 per cent of 149 cases were associated with pregnancy. A recently published series of 21 cases reported by Wells, Margolin,

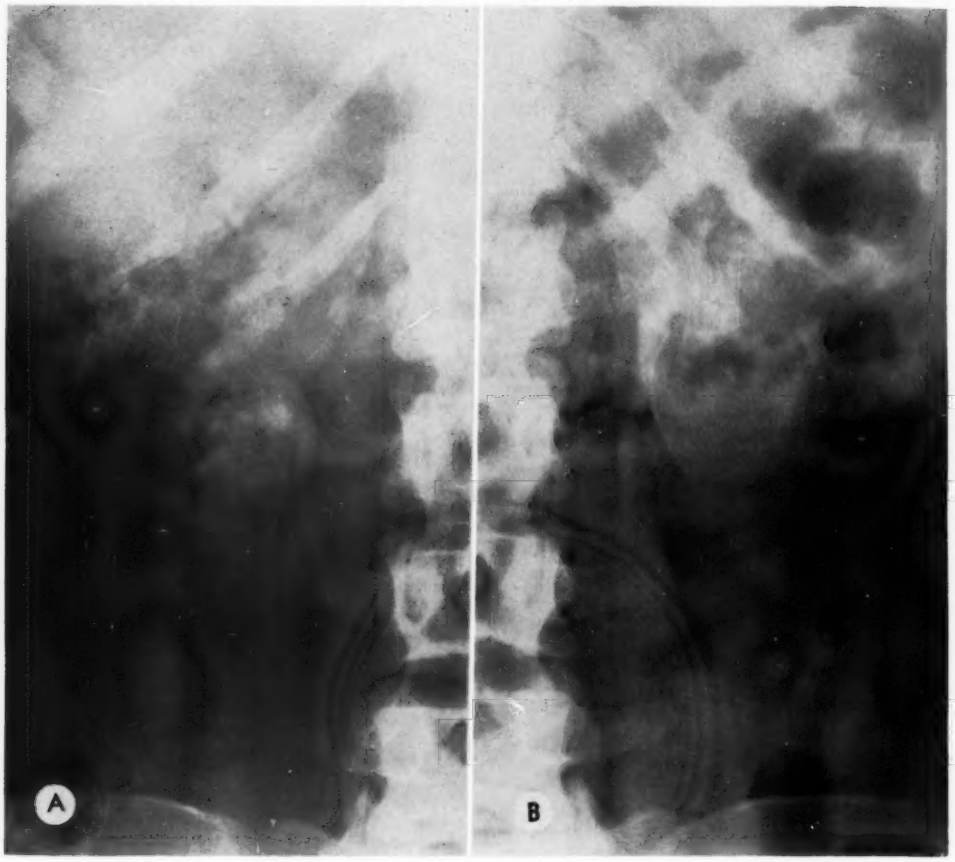

FIG. 3. (*A* and *B*) Intravenous pyelograms one and one-half years after the acute episode show prompt excretion bilaterally with only moderate concentration of the contrast material. The pelvicalyceal systems appear normal.

and Gall[20] is at variance with the surveys cited, in that only 14 per cent of their cases were preceded by the hemorrhagic complications of late pregnancy. No definite conclusion can be drawn from this relatively small group of cases. The more frequent use of needle biopsy in suspected cases of cortical necrosis may substantiate their implication that the high degree of association with pregnancy is more apparent than real.

No specific etiologic agent has been uncovered as the cause of cortical necrosis. However, renal ischemia is considered the pathogenetic factor of greatest importance in the production of the characteristic necrotic cortical lesions. The mechanism of ischemia, though extensively investigated, has not been satisfactorily explained. The theory of renal vasospasm has received the widest support. Davis feels that cortical necrosis is produced by transitory vasospasm of intrarenal arteries for periods of from perhaps two to four hours, followed by the restoration of blood flow.[5] No vasopressor substance has as yet been isolated from patients with cortical necrosis. The experimental production of vasospasm sufficient to cause cortical necrosis has centered about toxins, poisons, and vasoconstrictor substances.[13] Included among these are serotonin and mercuric chloride. Other investigators have implicated embolism, vascular shunts, vasoparalysis, vascular damage, and fibrin deposition as causative factors in the induction of renal ischemia.[7,8,13,18]

Although the entire renal cortex may be-

come necrotic, Duff and More[7] state that usually only its outer one-half to two-thirds is affected. A narrow rim of normal subcapsular parenchyma is often spared, presumably by reason of collateral capsular circulation. If the patient survives cortical necrosis, the recovery of renal function is more or less incomplete, in contradistinction to the complete restoration of function in acute tubular necrosis.[10] The outcome will depend to a considerable extent on the amount of tissue destroyed.[20] Even with extensive cortical necrosis, prolongation of life through meticulous care to the problems of fluid and electrolyte balance as well as by the use of hemodialysis has allowed sufficient time for the calcification of necrotic cortical tissue, as in the present case.

As stated earlier, a review of the literature disclosed 4 cases of renal cortical necrosis in which cortical calcification has been demonstrated roentgenographically in the living patient. The calcifications were first observed at seven weeks,[15] fifty-seven days,[6] two months,[2] and sixty-one days[11] after the onset of the acute episode. In our own case the cortical calcification was first seen on the fifty-seventh day. In DeGraeff and DeBaan's[6] case microscopic calcification was observed on a renal biopsy taken on the twentieth day and cortical calcification was seen on the plain roentgenograms of the abdomen on the fifty-seventh day.

In a case reported by Gjørup, Killmann, and Thaysen,[10] diffuse cortical calcification was found on a renal biopsy specimen obtained on the thirty-eighth day and a thick renal cortical calcific shell was observed at the time of necropsy on the fifty-first day. No roentgenograms had been obtained. According to Sheehan and Moore,[18] as well as other investigators, calcification in this condition is an uncommon finding at the time of autopsy. A review of 21 cases encountered at the Cincinnati General Hospital[20] failed to demonstrate renal calcification at necropsy. However, death occurred in these cases, as in a majority of those reported, within five to thirty-one days.

From the information available it appears that a prerequisite to the roentgenographic demonstration of cortical calcification in cortical necrosis is survival of the patient for at least one and one-half to two months. Mortensen *et al.*[16] have pointed out that there is not always a close correlation between the quantity of calcification visible microscopically and that demonstrated roentgenographically. In some instances the calcification demonstrated microscopically may not be visible on roentgenograms. Moëll[15] reported a case in which cortical calcification was seen on a postmortem roentgenogram of the kidneys but not on roentgen studies made prior to death.

DIAGNOSIS

The diagnosis of bilateral renal cortical necrosis is definitively made by biopsy or at the autopsy table.[4,17] Some investigators feel that there are clinical features of practical value in differentiating this type of acute renal failure from that occurring in acute tubular necrosis, acute glomerulonephritis, acute bilateral renal arterial or venous occlusion, and extensive bilateral renal infarction.[20] The frequent association of cortical necrosis with the hemorrhagic complications of late pregnancy is emphasized, as is persistent anuria or severe oliguria, gross hematuria at the onset, and flank pain early in the course of the disease. These manifestations, however, are not specific and may be found in association with other forms of acute renal failure, particularly acute tubular necrosis. Bilateral cortical necrosis should be suspected, however, when acute anuria occurs in a multiparous woman during the last trimester of pregnancy, when accompanied by vaginal bleeding, toxemia, or pre-existing hypertension. The disorder should be considered in acute renal failure in children, especially when diarrhea, vomiting, and dehydration are part of the clinical picture. The diagnosis should also be entertained in cases of acute renal failure associated with septic shock.[13] Although the history and clinical course is suggestive of the diagnosis, the

roentgenographic findings may substantiate this impression. Gjørup and his coworkers[10], along with Margolin[14], feel that the appearance of a rim of renal cortical calcification, when associated with a compatible history and clinical course, is diagnostic of bilateral cortical necrosis.

ROENTGEN FINDINGS

Early in the course of bilateral renal cortical necrosis the kidneys may enlarge. This is followed by a variable degree of renal shrinkage. In our case the shrinkage occurred primarily during the first few months following the acute episode. However, further reduction in the size of the left renal outline was noted on roentgenograms taken nineteen months later. The faint cortical calcification, which has the appearance of penciling-in of the kidney margins, appears about one and one-half to two months after the onset of the disease. Both an anatomic and a pathologic basis exist for the appearance of this type of calcification. The normal renal cortex measures 5 to 8 mm. in thickness. Since a subcapsular zone as well as approximately one-third to one-half of the inner cortex are usually spared of necrosis,[7] it follows that only a relatively thin intervening layer of necrotic tissue is susceptible to calcification. Irregularity of the renal contour as well as interruption of the cortical calcification may occur. This is a reflection of the patchy distribution of the necrosis. The faint calcifications may be difficult to see on conventional roentgenograms of the abdomen, and laminagraphy may demonstrate the calcification to better advantage (Fig. 2, *A* and *B*). Laminagraphy is, therefore, recommended in suspected cases of renal cortical necrosis. In our case intravenous pyelography nineteen months after the acute episode revealed prompt excretion bilaterally with only moderate concentration of the contrast material (Fig. 3, *A* and *B*). The pelvicalyceal systems appeared normal.

Calcification somewhat similar to that observed in renal cortical necrosis has been reported in chronic glomerulonephritis, and on postmortem kidney roentgenograms of a case in which there were widespread osteolytic metastases to bone.[3] The history will serve to differentiate these cases from cortical necrosis. A number of conditions and substances have been associated with the production of renal calcification.[1,3,12] Examples of these are hyperparathyroidism, milk-alkali syndrome, renal tubular acidosis, sarcoidosis, chronic pyelonephritis, sulfonamide tubular damage and others.[9] To our knowledge the nephrocalcinosis associated with these conditions has not been confined, roentgenographically at least, to the renal cortex, and therefore they do not offer a problem in the differential diagnosis of bilateral cortical necrosis.

SUMMARY

A case of bilateral renal cortical necrosis is presented. Certain etiologic, clinical, and differential diagnostic features of this condition are discussed. The literature on bilateral renal cortical necrosis has been reviewed with special attention directed to the appearance of a characteristic type of renal calcification detectable roentgenographically. The present paper stresses the importance of this calcification in the separation of bilateral renal cortical necrosis from other causes of acute renal failure.

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URETHROGRAPHY IN THE ADULT MALE*

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FOR the past ten years, the subject of adult male urethrography has received little attention in the American radiologic literature. Several papers have considered the newer contrast media,[3-7,11,14] and specific applications for adult urethrography have been reported,[9,12,13,15] but only a few general reviews of this subject have been published.[10,14,16] It is the purpose of this paper to review these general considerations and our experience with retrograde urethrography, and perhaps to stimulate more general acceptance and usage of this simple and valuable diagnostic procedure.

It must be emphasized that retrograde urethrography as described in this presentation is of value only in the determination of disease processes which produce anatomic change in the male urethra, and that the procedure by itself is of limited value in the study of the physiology or pathophysiology of the lower urinary tract. These latter processes are best examined by voiding cystourethrography, preferably with a rapid cassette changer or cinefluorography. We feel that the infant male urethra is also best examined roentgenologically by using voiding cystourethrography, although there are instances when the retrograde study in conjunction with the voiding study is necessary to establish the diagnosis in a young patient.

CONTRAST MEDIA

Since Cunningham performed the first contrast visualization of the urethra with argyrol in 1910,[1] there has been a continuing search for a satisfactory medium for urethrography. The use of aqueous contrast media is attended by very few complications, but these media are not sufficiently viscous to distend the posterior urethra for adequate visualization of this structure. The oily media possess the desired viscosity but instances of pulmonary oil embolism have been reported.[17] Barium-water suspensions are also unsuitable for retrograde urethrography.[8] Thixokon (Mallinckrodt) is a water soluble medium with sufficient viscosity to distend all portions of the urethra and we believe it is the medium of choice. We first began to use thixokon in 1956 on an investigational basis. Since that time almost 200 urethrographic examinations have been performed without a single instance of toxic reaction or side effect, although upon several occasions the medium has inadvertently been extravasated into the corpus spongiosum and pelvic veins.

A comparison of thixokon urethrograms with those made previously with other media shows that the thixokon studies, almost without exception, demonstrate the entire urethra, whereas it was often impossible to force other media through the external sphincter. This may be due to the fact that thixokon is less irritable and allows greater pressures to be applied to the sphincter.

It has been our experience that thixokon possesses greater radiopacity than other aqueous media and therefore produces roentgenograms of superior technical quality. This greater opacity may occasionally result in missing a small radiolucent filling defect within the urethral lumen; this disadvantage may be overcome by the use of high kilovoltage techniques.

PROCEDURE

The equipment necessary to perform retrograde urethrography is quite simple, and, with the possible exception of the Brodny[2] instrument, is readily available in

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any radiologic department. The procedure itself is performed in a period of time rarely exceeding twenty minutes. A preliminary survey roentgenogram is made and examined for positioning and technique. The Brodny instrument is then attached and with constant pressure 20 to 25 cc. of medium is *slowly* injected. With experience, it is possible to "feel" when the hydrostatic pressure of the medium has overcome the resistance of the external sphincter and allowed the medium to fill the posterior urethra. At this time, four roentgenograms are made using the anteroposterior projection, each oblique projection with the patient in the "bicycle riding" position, and an evacuation film after removal of the conical tip portion of the Brodny instrument. The patient is asked to void as this last exposure is made.

For several years, our urethrograms were made with fluoroscopic guidance, employing various pressure maneuvers in an attempt to study the physiology as well as the anatomy of the lower urinary tract. This practice has now been discontinued because it was found to add little to the diagnostic usefulness of the retrograde study. In addition, fluoroscopy made the procedure technically more difficult and increased the exposure to both the patient and the operator. With the present four film technique and the use of the Brodny instrument, the exposure to the operator's hands is less than 15 mr per examination. We feel that this small exposure does not warrant the use of cumbersome lead gloves, although the operator does protect his body with a fluoroscopic lead apron.

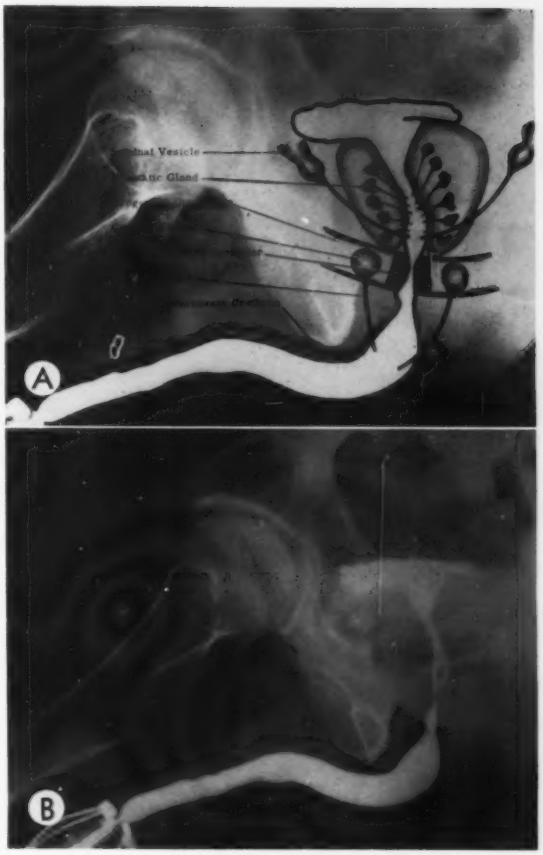

Fig. 1. (*A*) Superimposed diagram showing normal anatomy. (*B*) Normal urethrogram.

Review of Normal Anatomy

The male urethra can be considered to be divided into three parts (Fig. 1*A*). The posterior urethra is that part which is surrounded by the lobes of the prostate gland and for that reason it is frequently called the prostatic urethra. This segment of the urethra averages 2.5 to 3.0 cm. in length and in cross section is crescent-shaped. The posterior elevation is produced by the verumontanum which, together with the urethral crests, extends the entire length of the posterior urethra. The seminal vesicles extend obliquely through the lateral lobes of the prostate and empty as the ejaculatory ducts on the apex of the verumontanum. On either side of the veru are the orifices of the ducts which drain the numerous prostatic glands.

The membranous urethra is that segment which lies within the layers of the urogenital diaphragm. Surrounding this part of the urethra is the external urinary sphincter. Cowper's glands are also located within the urogenital diaphragm, although the ducts from these glands enter the urethra in its bulbous portion.

The anterior urethra extends from the external urinary sphincter to the urethral meatus and is surrounded for almost its

FIG. 2. Uncomplicated anterior urethral stricture of venereal origin. The irregularity and lack of distensibility of the entire anterior urethra indicate chronic infection. A second, large-caliber stricture is present.

entire length by the corpus spongiosum. The anterior urethra is subdivided into the bulbous, or fixed, portion and the penile portion distal to the penoscrotal junction. Numerous small submucosal glands empty into the anterior urethra along its distal two-thirds.

Figure 1*B* illustrates the normal appearance of the urethra as seen on the urethrogram. The verumontanum produces a radiolucent filling defect in the posterior urethra. The membranous urethra is the narrowest part and is demarcated by the more distensible portions of the urethra just proximal and distal to it. The anterior urethra is normally smooth in contour and quite distensible. The area of narrowing just distal to the membranous urethra is called the musculus compressor nudae[10] and must not be mistaken for the membranous urethra itself or for an anterior urethral stricture. In the normal individual, none of the glandular structures or their ducts are visualized.

INDICATIONS

In general, the indication to us for retrograde urethrography in the adult male is the presence of any lower urinary tract symptoms which are not readily explained by the usual diagnostic modalities of history, physical examination, laboratory studies, and endoscopy. Some authors advocate urethrography for all adult males with lower urinary tract symptoms and we are in agreement with their opinion. Unfortunately, we have found that, in our hospital, routine urethrography is not feasible for both administrative and economic reasons and we have had to limit the study to those patients in whom some diagnostic difficulty exists.

The specific indications for urethrography will be considered separately with illustrative cases.

ILLUSTRATIVE CASES

CASE 1 (Fig. 2). This fifty-six year old male had been experiencing frequency, nocturia, dysuria, and dribbling for many years. These symptoms had been progressing in severity and just before urethrography was performed he had been admitted to the hospital in acute urinary retention. The admission diagnosis was acute retention secondary to prostatic hypertrophy. When attempts to catheterize the patient were unsuccessful, a urethrogram was obtained. Upon later questioning, the patient admitted social indiscretions as a youth.

Comment. Urethrography is indicated in all cases of urethral stricture because it not

FIG. 3. Multiple anterior urethral strictures of venereal origin complicated by a urethrocutaneous fistula. Note the other stigmata of anterior stricture: dilated, infected Cowper's ducts and prostatic glands; false passages from attempted dilatations; contracted vesical neck caused by chronic infection.

only establishes the diagnosis of stricture but also demonstrates the location, number, and extent of the strictures, as well as the presence or absence of any of the complications of urethral stricture.

Case II (Fig. 3). This sixty-nine year old male had been treated for many years with periodic dilatations for urethral stricture. Shortly before this urethrogram was made, the patient developed a perineal abscess which promptly resulted in a urethrocutaneous fistula. Upon admission to the hospital, he was found to be unable to pass urine through the urethral meatus but was voiding without difficulty through the perineal fistula. The patient is now well following urethroplasty, suprapubic prostatectomy, and Y-plasty of the vesical neck.

Comment. Urethrography is necessary in all patients with urethral fistulae to determine the etiology of the fistula as well as to aid in planning the proper surgical approach. This study is also the only available method for examining the urethra proximal to the most distal urethral stricture present.

Case III (Fig. 4). This forty-eight year old male had had two transurethral resections prior to this urethrogram. These had been performed twelve and five years previously but had failed to relieve the symptoms of frequency, urgency, and nocturia. Proper resection of the previously unresected adenoma provided complete relief of symptoms.

Fig. 4. Residual prostatic tissue following incomplete transurethral resection. The unresected adenoma is exerting a ball-valve effect as it lies within the prostatic fossa.

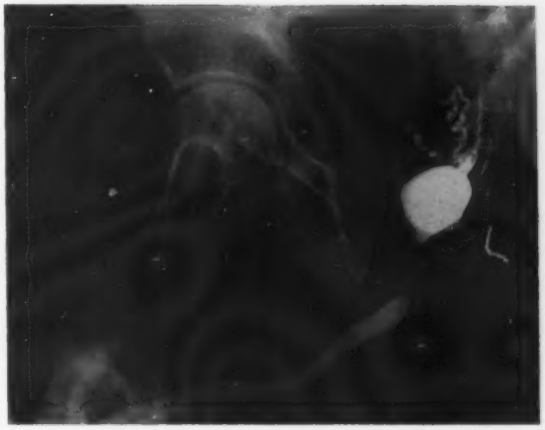

Fig. 5. Sclerosis of the bladder neck following transurethral resection. Note the balloon-like dilatation of the prostatic fossa caused by the retrograde injection of opaque medium. The seminal vesicles are filled and one large false passage is present.

Case IV (Fig. 5). This sixty-nine year old male had had a transurethral resection for symptoms of prostatism five years prior to this study. He was symptomatically improved for several months following the resection. The frequency and nocturia then recurred and gradually became more severe than they had been before his operation. A Y-plasty of the contracted, sclerotic bladder neck was planned, but the patient refused further surgery and expired in uremia.

Comment. Cases III and IV demonstrate the value of urethrography in the post-prostatectomy patient who continues to have obstructive symptoms. We believe that all such patients should have urethrography performed before a second resection is attempted.

Case V (Fig. 6). This fifty-two year old male suddenly developed profuse, painless urethral bleeding one week before this study was done. His urinary function was not impaired and he denied any previous urinary symptoms. Endoscopy after spontaneous cessation of the bleeding revealed no abnormalities. The urethral diverticulum was excised through a perineal incision and the patient is again asymptomatic.

Comment. Urethrography is indicated in all patients with urethral bleeding and no

FIG. 6. True (congenital) urethral diverticulum. The urethral bleeding was apparently caused by rupture of the distal end of the diverticulum into the bulbous urethra. The proximal connection between the diverticulum and the urethra was not demonstrated by this study. At surgery, this connection was found just proximal to the membranous urethra.

FIG. 7. A well-formed, large caliber, anterior urethral stricture six weeks following definitive repair of a severed urethra.

history of recent trauma, although it is well to wait until the bleeding has subsided before attempting the procedure. In this patient, neither of the orifices of the diverticulum was visualized by endoscopy and the diagnosis was in doubt until urethrography was performed.

CASE VI (Fig. 7). This five year old male child fell astride a bicycle injuring the perineum just dorsal to the scrotum. Immediately after the injury, there was gross urethral bleeding and he was unable to void. The urethra was surgically repaired over a splinting catheter passed through a cystotomy incision. This urethrogram was made six weeks following injury and operation at which time the patient was asymptomatic, although a well-formed stricture is already present.

Comment. Urethrography is a routine part of the postoperative management of urethral injuries resulting from external trauma. The study demonstrates the presence or absence of stricture formation at that time and serves as a baseline for further studies should the patient develop obstructive symptoms in the future.

CONTRAINDICATIONS

Contraindications to retrograde urethrography are few. It is our opinion that acute urethral injury from external trauma is one of these contraindications because the diagnosis is evident by history and physical examination alone and because the extravasated opaque medium may in itself contribute to stricture formation. In the unusual case of this kind in which urethrography may be necessary, an aqueous contrast medium should be used.

It has been our experience that most urethrograms made shortly after instrumentation of the urethra were not of diagnostic quality due to extravasation of the contrast medium through small mucosal tears resulting from the previous internal trauma. The study is therefore best performed before attempting catheterization or endoscopy.

Urethrography should be postponed in the presence of acute infection because of the possibility of producing vascular dissemination of the infecting organisms.

SUMMARY

We believe that urethrography is a much neglected but quite useful diagnostic procedure. It is hoped that this review will stimulate increased interest in and applica-

tion of this method for investigation of the lower urinary tract.

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LIPOMA OF THE CORPUS CALLOSUM*

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LIPOMA of the corpus callosum is a rare intracranial midline lesion. The first pathologic description of this entity was presented by Rokitansky.[26] Other reports have since appeared in the literature.[2,3,9,12,19,25,27,32,34] We have been able to find only 13 out of about 50 previously reported cases that had been correctly diagnosed prior to operation or death. The first roentgen diagnosis on plain skull roentgenograms was made by Sosman[31] in 1939.

The purpose of this paper is to present an additional case of lipoma of the corpus callosum which was diagnosed on plain skull roentgenograms prior to death and to show the distribution of calcification in the lesion with the aid of roentgenograms of brain slices.

PATHOGENESIS

The most commonly accepted hypothesis of the pathogenesis of lipoma of the corpus callosum is that of Krainer,[20] and of Wassermann.[36] They believe that lipoma develops from persistent primitive meningeal tissue, and state that this occurs in the leptomeningeal cisterns. This hypothesis was concurred in by Merkel,[22] List *et al.*,[21] and Vonderahe and Niemer.[35] These authors assume that the development of lipoma occurs in very early embryonic life (about the second fetal month) and persists into maturity.

The interference with the development of the corpus callosum and the adjacent structures by the persistence of this primitive tissue has been discussed by de Morsier.[23] It seems very likely that agenesis, or partial agenesis, of the corpus callosum is the consequence of this congenital lesion. In our opinion the interference with development is dependent upon the location of the persistent primitive tissue. Our belief is based on the fact that the splenium and septum pellucidum are absent in the presence of a lipoma, as seen in our case. Similar findings were reported by Würth,[38] List *et al.*,[21] and Huddleson,[15] who observed total agenesis of the corpus callosum, and Juba,[17] Huebschmann,[16] Rubinstein,[27] von Sury,[32] Benjamin,[2] Ehni and Adson,[11] Schnürer and Zettergren,[28] Kirkbride,[19] Dyke,[10] Ernst,[12] and Sheinmel and Lawrence,[29] who were able to demonstrate partial agenesis of the corpus callosum in the presence of lipoma. Sosman[31] also reported agenesis of the corpus callosum in his 3 cases.

A few cases are described by Wolfson *et al.*[37] without associated congenital anomalies. In these cases the lipoma was situated above the corpus callosum, tapering in anteroposterior direction. These various findings support the belief that the original localization of the primitive tissue is mainly responsible for associated anomalies, and that one is dealing with a secondary agenesis of the corpus callosum. The latter must be clearly distinguished from primary agenesis, as shown in 2 case reports by Sheinmel and Lawrence[29] and Zellweger.[39] Baker and Graves[1] did not find any lipoma in their 82 cases of partial agenesis of the corpus callosum, nor did Bunts and Chaffee[7] in their 15 cases.

Lipoma of the corpus callosum is a benign tumor composed of mature fat and fibrous tissue and resembles the typical lipoma of any other region of the body. It is not infiltrative and does not possess atypical cells or abnormal mitoses.

Lipoma occurs with equal frequency in the male and female. The age distribution

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varies from thirty months[24] to seventy-five years.[13]

Lipoma of the corpus callosum may be asymptomatic. Signs and symptoms, when present, are those of a midline lesion of the brain and are not specific for lipoma. Persistent severe headaches, grand mal seizures, hemiparesis and mental changes have been variously reported.[5]

A definitive diagnosis of lipoma of the corpus callosum can be made, during life, from the characteristic roentgenographic appearance.[21]

REPORT OF A CASE

A seventy-one year old white male (Unit No. C-58420) was admitted on October 26, 1959 with a chief complaint of persistent headaches since 1944, associated with occasional dizzy spells and weakness of legs. The positive physical findings were limited to the central nervous system and indicated a spinal cord lesion. Myelography revealed a block at the level of the eighth and ninth dorsal segments. The block was considered to be secondary to a metastatic tumor. Complete clinical data of this patient will appear in another report.[33]

Roentgenograms of the skull were taken on admission. In the posteroanterior view there were symmetric parasagittal curvilinear densities with lateral convexities. The distance between these densities measured about 2 cm. (Fig. 1*A*). A curvilinear density with superior convexity was observed in the lateral view in the region of the corpus callosum. Inferior to this was a zone of relative radiolucency (Fig. 1*B*). A diagnosis of lipoma of the corpus callosum was made (P.B.W.). A repeat roentgenographic examination of the skull was performed about two weeks later and revealed the same findings.

Pneumoencephalographic and arteriographic studies were not performed because of the patient's general condition.

The patient was treated symptomatically and was discharged to a nursing home on November 27, 1959 and died there on December 16, 1959.

Necropsy revealed that the patient had a bronchogenic carcinoma with metastasis to the spinal dura, producing the block and cord compression that resulted in a cord bladder, complicated by suppurative pyelonephritis. For the purposes of the present report we will limit our remarks to the description and discussion of the brain.

Roentgenograms of the formalin-fixed brain were made prior to dissection so as to visualize the ventricular system without the benefit of air injection. There were dilatation of the posterior horns of the lateral ventricles, more so on the left, and marked separation of the frontal horns. Calcification within the tumor was seen in various projections.

The brain was dissected after ten days' fixa-

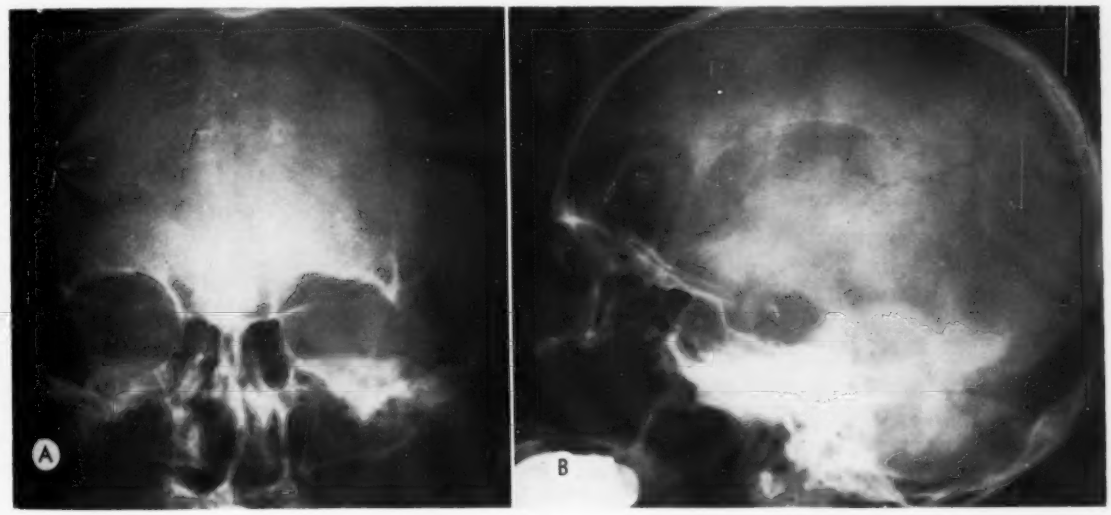

Fig. 1 (*A*) Posteroanterior roentgenogram of skull. Symmetric curvilinear densities with lateral convexities are present in a parasagittal position. (*B*) Lateral roentgenogram of skull demonstrates the curvilinear density with the superior convexity and the relatively radiolucent area, inferior to the dense line.

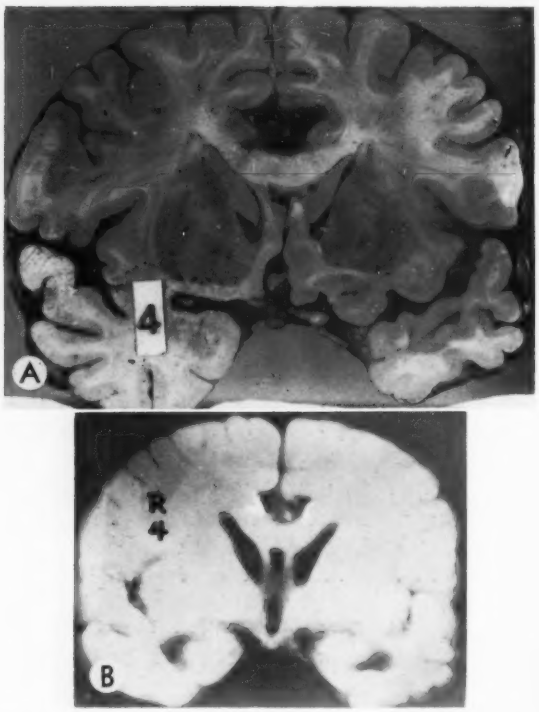

FIG. 2. (*A*) Coronal section and (*B*) roentgenogram through midportion of anterior horns of the lateral ventricles. There is definite dilatation of the sulcus corporis callosi. The septum pellucidum is absent and the fornices columnae are attached to the corpus callosum. At this level, the lipoma is only a thin layer over the corpus callosum. The calcified bands accompany the tumor laterally into the adjoining brain substance.

tion in 10 per cent formalin. It weighed 1,600 gm. The gyri were flattened and the sulci were slightly widened. The cerebral hemispheres were equal and symmetric. The cranial nerve distribution was normal. The arteries were in normal position. They were focally narrowed by atheromatous plaques. The pia mater was thickened and opacified.

There was an irregular, lobulated, yellow, fatty tumor involving the corpus callosum.The main body of the tumor within the corpus callosum measured 2.7×3.1×2.5 cm. There were several offshoots extending in various directions. One of these was a sheet, 1.9 cm. wide and 0.2 cm. thick, covering the surface of the corpus callosum from the genu to a point approximately 7 cm. posterior to the genu. This tissue covered the raphe, the striae longitudinales mediales and laterales and a small portion of the gyri cinguli. Another projection presented in the right lateral ventricle as a bulbous mass 1.2 cm. in diameter connected to the main mass by a stout pedicle occupying a dilated foramen of Monro. Still another projection of tumor presented as a cap over the third ventricle. The anterior cerebral arteries were thickened and were contained within the tumor tissue. The tumor appeared to be well defined, with a fine membranous capsule and was soft except for numerous discrete and confluent hard white calcified plaques. Some of the latter presented as tiny flecks in the capsule, while more concentrated masses were found in the superior portion of the corpus callosum and in the striae longitudinales laterales. There were anomalies of the corpus callosum, including incomplete development of the splenium, absence of the septum pellucidum and widening of the sulcus or cisterna corporis callosi. The cut surfaces of the corpus callosum presented a watered silk appearance caused by interlacing bands of pale yellowish-white fibrous tissue.

Photographs and roentgenograms of 8 coro-

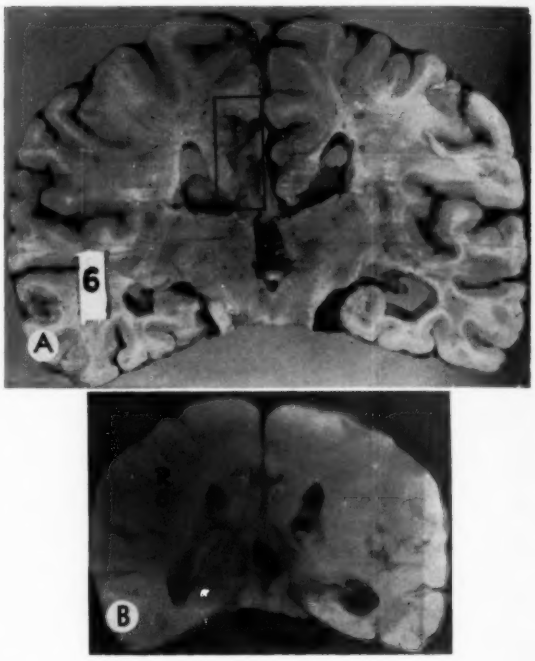

FIG. 3. (*A*) Coronal section and (*B*) roentgenogram through posterior third of body of lateral ventricle. The continuity of the corpus callosum is interrupted by the main mass of the lipoma. A projection of the tumor forms a cap over the dilated third ventricle while another projection extends into the right lateral ventricle. The fibrous tissue streaks in the corpus callosum are readily seen. *B* shows that the visible calcium still appears as a thin band adjacent to the sulcus corporis callosi. The rectangle outline in *A* indicates the location from which the section shown in Figure 4*A* was taken.

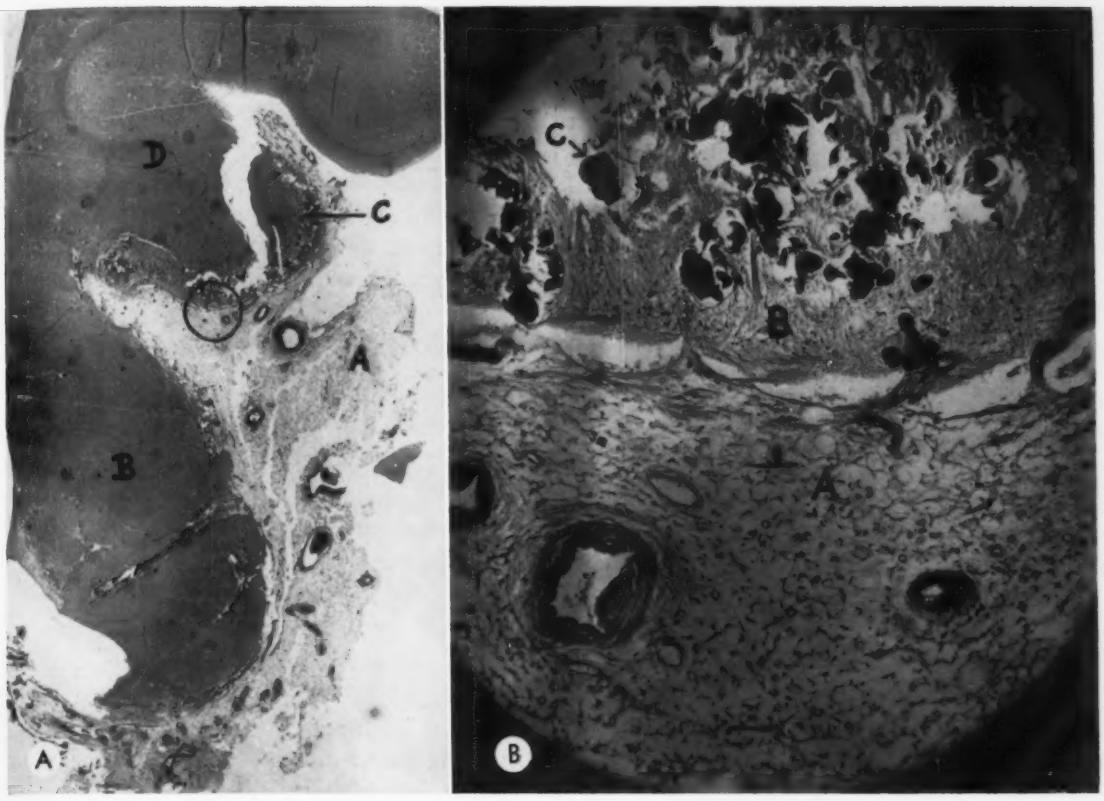

Fig. 4. (*A*) Low power view (5×) of rectangular section of Figure 3*A* shows adjacent corpus callosum and gyrus cinguli. A, lipoma; B, fragment of corpus callosum; C, calcification; D, gyrus cinguli. The area bounded by the circle is shown in *B*. (*B*) This demonstrates the mature adipose tissue of the lipoma with numerous thick-walled vessels. Note the deposition of calcium in the adjacent brain tissue. It is this calcification which is seen on roentgenograms (80×). A, fat tissue; B, brain tissues; C, calcification.

nal brain sections containing the tumor were taken. Only numbers 4 and 6 are included in this paper (Fig. 2, *A* and *B;* and Fig. 3, *A* and *B*). The others exhibited similar features.

Microscopic study showed the tumor composed of mature fat cells with abundant vascular support in the periphery and with fine collagenous stromal support producing incomplete lobulation. The continuity of the corpus callosum was interrupted, as previously described. The intermediate space was occupied by this same fatty tissue, in some fields bounded by coarse bands of dense collagenous tissue. The latter occurred also in irregular bands on the surface of the corpus callosum and within the corpus callosum, mostly around blood vessels. It is this tissue which was responsible for the coarsely trabeculated irregularities in the cut surface of the corpus callosum. Small and large calcified bodies were found mainly in the periphery of the fatty tumor and in the adjacent brain tissue (Fig. 4, *A* and *B*).

DISCUSSION

The roentgenographic features of lipoma of the corpus callosum are very characteristic but there is no uniformity in the reported location of the calcifications seen on the plain roentgenograms.

It has been variously stated that the calcifications are present in the tumor, in the capsule of the tumor, in the adjacent brain tissue, or in all of these locations simultaneously. This statement is certainly true from a histopathologic standpoint. However, the characteristic calcifications which are identifiable on the plain skull roentgenograms are chiefly located in the brain tissue bordering the sulcus corporis callosi, as seen in our case. The size and the concentration of the visible calcification are not related to the size of the tumor. The latter is variably described from pea-size to egg-size or as a

sheet spread over the surface of the corpus callosum. The pressure of the tumor on the highly sensitive adjoining brain substance affects the circulation. The amount and the intensity of calcium deposit are dependent upon the degree of circulatory disturbance. This conclusion has been arrived at by Bull.[6]

Roentgenograms of the individual coronal brain sections show calcific densities which produce the parenthesis-like bilateral densities visualized in the posteroanterior view of the skull. The continuity of the calcification in the anteroposterior direction along the superficial brain substance of the gyri cinguli is demonstrated on the lateral roentgenograms of the total brain.

We assume that the visible calcifications are, in most instances, in the adjoining brain tissue because of the uniformity of the characteristic roentgenographic features of published roentgenograms of cases of lipoma of the corpus callosum. The distance between the parenthesis-like calcification in the posteroanterior view depends upon the lesser or greater widening of the sulcus corporis callosi. Another observation favoring our hypothesis is the fact that the curvilinear density in the lateral view constantly appears with a convexity directed superiorly, and it has never been demonstrated with the convexity directed inferiorly.

The second pathognomonic sign, the so-called radiolucent area inferior to the curvilinear density in the lateral view, depends upon the size of the compact fatty tumor mass. This area may be a true radiolucency or a lesser degree of density in comparison to the surrounding brain.

Diagrams of plain roentgenograms of the skull of reported cases of lipoma of the corpus callosum are presented by Mullen and Hannan,[24] who noted the constancy of these calcified lines and of the radiolucency in some cases. The same is true in our case.

We would emphasize that no other intracranial midline lesion presents a similarly demarcated radiolucent zone and such typical curvilinear calcification. It is possible, thereby, to distinguish lipoma from primary agenesis of the corpus callosum, congenital cyst of the cavum septi pellucidi and tumors of the corpus callosum such as astrocytoma, oligodendroglioma and ependymoma, named in the order of their frequency.

Successful removal of the total lesion has not been reported.[4,8,14,18,21,24,36] Occasional attempts have been made to excise the benign tumor but had to be terminated because of bleeding due to the vascularity of the lipoma and the envelopment of the anterior cerebral arteries and their branches by the tumor.

The patient's condition and symptoms should and can be very well controlled by sedatives and anticonvulsants.[30]

SUMMARY

1. A case of lipoma of the corpus callosum has been presented.

2. The diagnosis was established prior to autopsy by plain roentgenograms of the skull.

3. Evidence has been presented to show that this is a congenital malformation.

4. This is the first case reported in which roentgenograms of the coronal brain sections were taken to localize the calcium distribution.

5. A brief review of the history, pathogenesis, pathology and differential diagnosis is presented.

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ROENTGENOLOGIC FEATURES OF NEUROFIBROMAS INVOLVING THE GASSERIAN GANGLION*

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NEUROFIBROMAS arising from the intracranial portion of the fifth nerve are rare. Approximately 43 cases had been reported in the literature prior to this series. The present report is a consideration of the roentgenologic features in the 13 cases reported by Olive and Svien[3] in 1957, plus another case encountered since; an additional 12 cases were reported by Schisano and Olivecrona[4] in 1960. Because such a study is necessarily retrospective in character, it should not be implied that the manifestations described are pathognomonic of this lesion. However, some interesting features occur that, when considered with the clinical evaluation of the patient, should strongly suggest the presence of a neurofibroma in this region.

These tumors of the trigeminal nerve exist primarily within the middle or the posterior cranial fossa or both fossae. Histologically, they resemble neurilemmomas of the acoustic nerve. Jefferson[1] pointed out that most tumors involving the gasserian ganglion actually arise from the pharynx and invade through the base of the skull, as judged by the clinical history or the presence of metastasis to the cervical lymph nodes or both. All such lesions have been excluded from this series, and pathologic confirmation of each tumor has been established.

The importance of the correct interpretation of the roentgenologic features arises from the paucity of initial clinical signs referable to involvement of the fifth nerve in many cases. Thus, in some instances, an earlier diagnosis of trigeminal neurofibroma may be suggested by roentgenologic means.

FINDINGS IN PRESENT STUDY

In this study, the roentgenograms and clinical records of 14 patients seen at the Mayo Clinic who had solitary neurofibromas of the trigeminal nerve were reviewed. None of these patients had any evidence of Recklinghausen's neurofibromatosis. The tumors were located primarily in the middle cranial fossa in 7 patients, in the middle and posterior fossae in 3 and in the posterior fossa in 4. Roentgenographic changes were not present in one patient in each category.

In one other patient, a tumor 2.5 cm. in diameter was an incidental finding at necropsy; roentgenograms of the head had not been made, but there was no evidence of bony erosion.

FIG. 1. Decalcification of the greater wing of the sphenoid on the right, with severe medial destruction of petrous portion of right temporal bone.

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† The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

The bony changes accompanying the lesions in the middle fossa were those which might be expected to be associated with parasellar masses that expand slowly, and these changes corresponded to that portion of the middle fossa in which the tumor attained its maximal size. In 2 instances, the forward extension of the tumor resulted in decalcification of the greater wing of the sphenoid adjacent to the superior orbital fissure (Fig. 1). In another case, an extremely large tumor extended throughout the floor of the middle fossa medially and, therefore, eroded the side of the sella turcica and the anterior clinoid process as well as the base of the skull in the region of the foramen ovale; erosion of the optic canal and the medial portion of the petrous tip also was present (Fig. 2, *a* and *b*). Two other large tumors were associated with erosion of the side of the sella turcica, including the posterior clinoid processes. Two patients gave evidence of a soft-tissue tumor in the region of the pterygoid fossa and posterior nasopharynx (Fig. 3).

Fig. 3. Large soft-tissue mass extending into nasopharynx on right. The bony floor of the right middle fossa is destroyed. The shadow of a hairpin is superimposed over this region.

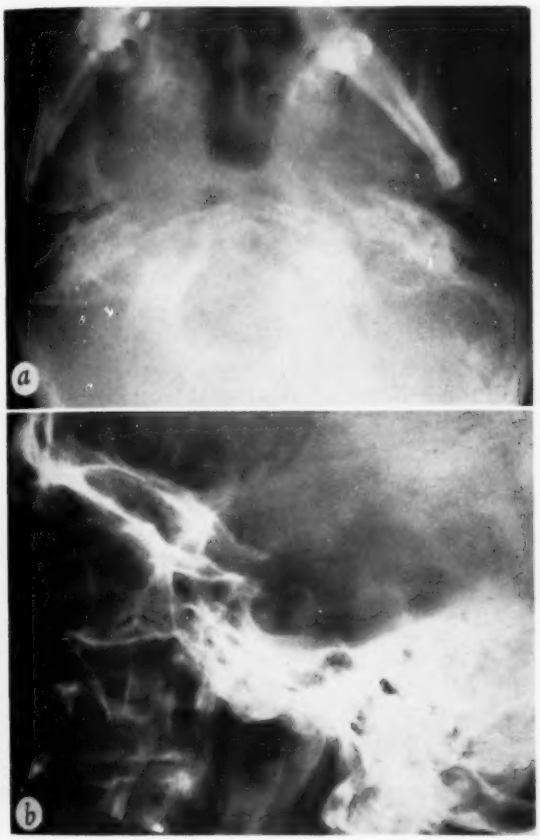

Fig. 2. (*a*) Extensive destruction of floor of right middle fossa, including right petrous tip. (*b*) Lateral view in same case, showing erosion of inferior aspect of right anterior clinoid and region of optic canal.

The most common and most significant osseous change involved the tip of the petrous bone. Erosion of the medial aspect of the petrous tip, leaving a rather sharp medial border, was noted in all 8 cases in which the petrous bone was involved (Fig. 4*a*). In each case, the destruction was medial to and did not involve the internal acoustic meatus. Although it has been reported that such destruction usually involves the superior portion of the tip, this was conspicuously lacking in at least one of our cases (Fig. 4*b*).

The floor of the middle fossa was known to be destroyed in 2 of the 3 cases in which the tumor was located partly in the middle fossa and partly in the posterior fossa. Erosion of the tip of the petrous bone medial to the internal acoustic meatus was

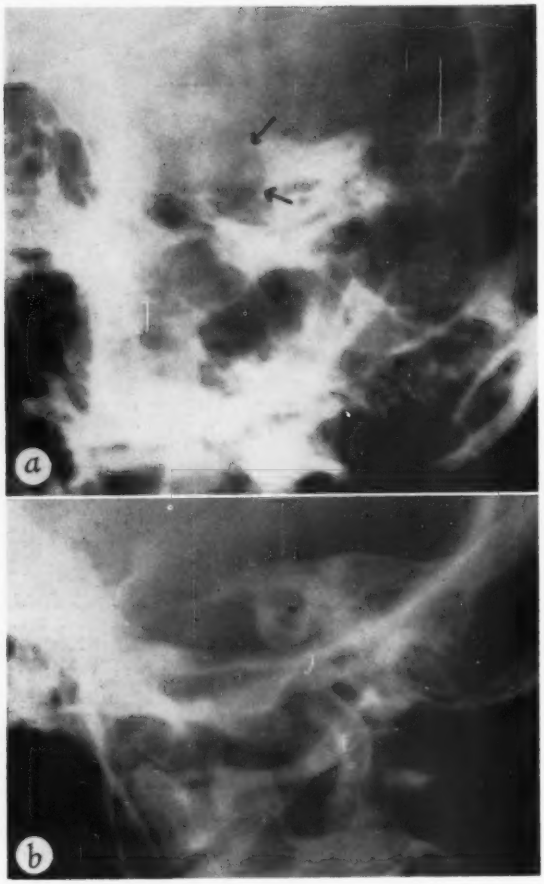

FIG. 4. (*a*) Destruction of left petrous tip medial to internal acoustic meatus, with well-defined, sharp, medial border (arrows). (*b*) Destruction of left petrous tip, with sparing of superior aspect.

present in both of these instances. These tumors may show the characteristics of growth in either of these cranial fossae. In those instances in which the bulk of the tumor was in the middle fossa, the findings previously noted prevailed.

When the mass was almost entirely within the posterior fossa, the roentgenologic characteristics were similar to those of a tumor at the cerebellopontine angle. However, as already noted, erosion of the petrous tip uniformly occurred medial to the acoustic meatus, which is not the usual feature accompanying neurofibromas of the eighth nerve. The rather sharp outline of the petrous tip at the site of the erosion helps to distinguish this lesion from the usual type of meningioma in the cerebellopontine angle. The appearance is not unlike that sometimes associated with erosion from an epidermoid tumor (Fig. 5).

Special Contrast Examination. Cerebral angiography was undertaken in 2 cases because the changes involving the sella turcica and parasellar region suggested the presence of a parasellar aneurysm of the internal carotid artery. In one of these studies, the internal carotid artery appeared to be compressed as it emerged from the region of the carotid foramen, suggesting compression by a mass immediately adjacent to it (Fig. 6). Both patients had some elevation of the vessels of the sylvian complex on the affected side, which indicated a mass in the middle fossa. It was our original impression on review of this material that the basilar vein of Rosenthal might have been elevated significantly. However, after a comparison was made with a number of angiograms in which lesions in this region were not present, it is our opinion that the basilar vein has a somewhat variable location and that it appeared to be in about the same position in many other cases that it was in these 2 instances.

Ventriculography was undertaken in one patient, who had a mass in both the middle and posterior fossae. Elevation of the temporal horn with amputation of its tip was demonstrated (Fig. 7). In addition,

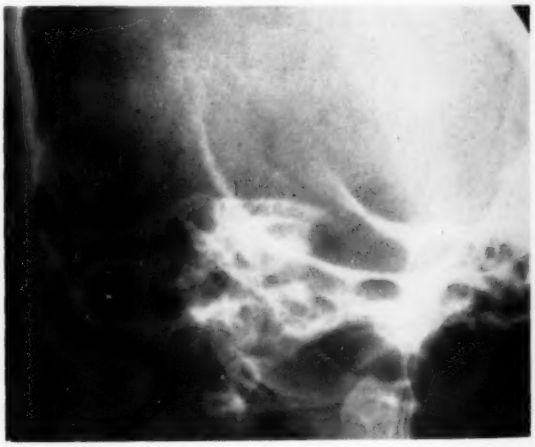

FIG. 5. Similar destruction of left petrous tip from epidermoid tumor.

Fig. 6. Medial compression of left carotid siphon and elevation of superior portion of siphon and middle cerebral artery by large parasellar mass.

this patient showed some dislocation of the aqueduct to the opposite side, together with evidence of obstruction of this channel.

Pneumoencephalography was undertaken in a patient whose tumor was at the cerebellopontine angle. Although the lesion

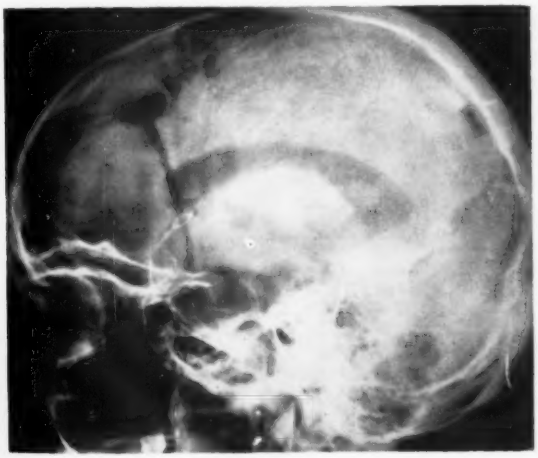

Fig. 7. Elevation and amputation of left temporal horn from recurrent large tumor in middle and posterior fossae.

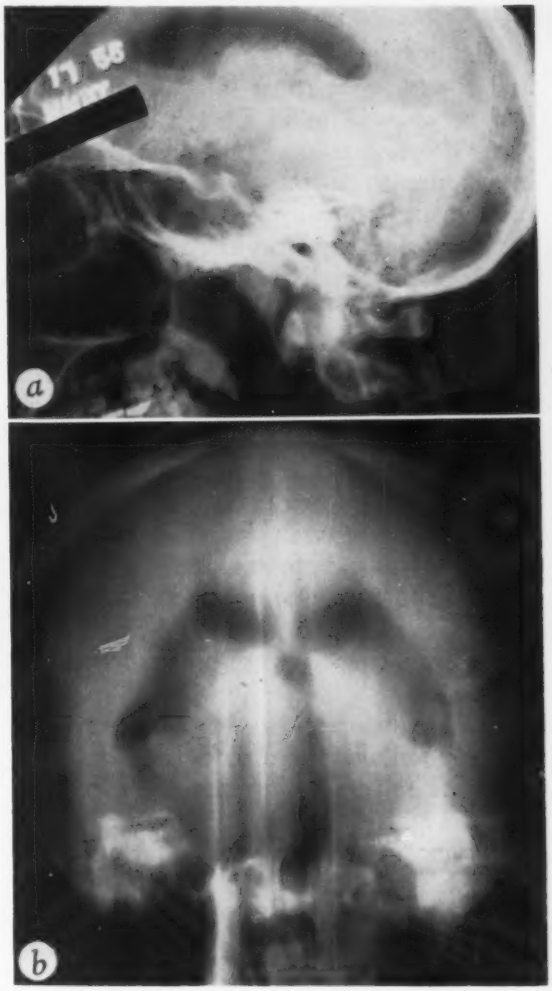

Fig. 8. (*a*) Right lateral pneumoencephalogram. A mass is displacing the aqueduct and fourth ventricle posteriorly. (*b*) Posteroanterior laminagram in same case, showing aqueduct and fourth ventricle displaced to right. Note destruction of the left petrous tip, with a sharp border medial to the internal acoustic meatus.

could be seen in the regular pneumoencephalogram, it was evident by means of laminagraphy that the aqueduct and fourth ventricle were displaced to the opposite side and that the aqueduct was displaced posteriorly by a mass compressing this portion of the brain (Fig. 8, *a* and *b*).

COMMENT

Previous authors have described some roentgenographic features associated with neurofibromas of the trigeminal nerve. In

1955, Jefferson[1] stressed regular but considerable enlargement of the foramen ovale and the foramen rotundum and erosion of the petrous bone as important findings. None of our patients had obvious isolated involvement of the foramen rotundum roentgenographically or at operation. Regarding pneumoencephalography, Lindgren[2] stated that displacement of the homolateral temporal horn was likely to be found in tumors of the fifth cranial nerve confined to the middle cranial fossa, and that extension of the tumor downward into the posterior fossa would produce evidence of hydrocephalus and displacement of the aqueduct and fourth ventricle. Lindgren concluded that carotid arteriography can afford only an approximate idea of that part of the tumor situated in the middle fossa of the skull, and that its usual value is in eliminating the possibility of an aneurysm.

Little mention apparently has been made of the possibility of a soft-tissue mass located in the pterygoid fossa and extending into the posterior pharyngeal soft tissues. Such a mass is common when the gasserian ganglion is involved by a metastatic process. It is important to note, therefore, that the presence of a retropharyngeal soft-tissue mass does not exclude the possibility of neurofibroma of the fifth nerve.

It is evident that the bony changes usually are related to the slow expansion of a mass and not to bony invasion by a neoplastic process, although the tumor in one case of our series grew through the petrous bone and presented in the middle ear and external auditory canal. Therefore, the changes observed are not those usually seen with other lesions in this region, such as chordomas, metastatic tumors, chemodectomas and pyoceles.

In general, these tumors attain a rather considerable size before disabling symptoms are observed. This may be related to the fact that the tumors arise from the nerve sheath and do not directly involve the functioning elements of the nerve until the lesions surround or expand them. This is different from the situation in which the gasserian ganglion is involved by a metastatic tumor. The chief differential diagnostic possibilities to be considered when the lesion is primarily in the middle fossa include aneurysm of the internal carotid artery and parasellar extension of an intrasellar adenoma. In most instances, this differentiation can be made by angiography.

By far the most characteristic single roentgenologic finding appears to be the type of erosion of the tip of the petrous bone. It should not be implied that this is the only pathologic process to produce such a sharp cutoff of the petrous tip medial to the internal acoustic meatus, but it is rather striking that this change was apparent in all 8 cases in which the petrous tip was involved. In most cases of bony involvement by a cerebellopontine angle tumor caused by other lesions, the changes are not so clear cut. When this change in the petrous tip is not present, there is little possibility of distinguishing this neurofibroma from one arising from the eighth nerve, which may not change the petrous bone. If it is remembered that most neurofibromas of the eighth nerve first alter the borders of the internal acoustic meatus, the presence of isolated destruction of the petrous tip might alert one to check carefully for localized changes in the middle fossa as well, in order to recognize a fifth-nerve neurofibroma that may have components in both the middle and posterior fossae. As study of the clinical features in these cases suggests, it is not possible always to make this distinction on a clinical basis. None of our patients showed any erosion of the internal acoustic meatus itself.

Erosion of bony structures within the middle fossa was observed in more than half of our patients. This varied from decalcification around the foramen ovale to extensive destruction of the floor of the middle fossa and the sella turcica. These tumors may cause considerable destruction of the middle fossa without extending very far into the soft tissues below.

Decalcification of the sella turcica from

increased intracranial pressure was encountered in 3 instances. This, of course, also is associated with other growths, particularly those causing obstruction to the ventricular system.

SUMMARY AND CONCLUSIONS

A study was made of the roentgenologic features in 14 cases at the Mayo Clinic in which neurofibromas involved the gasserian ganglion. Primary neurofibromas of the gasserian ganglion occur in the middle or the posterior fossa or both. They are rare and may present a problem in diagnosis because of a paucity of initial symptoms referable to involvement of the fifth cranial nerve.

The most characteristic roentgenologic feature is sharply circumscribed erosion of the petrous tip medial to the internal acoustic meatus. This change occurred in 8 of the 14 cases. Destructive changes in the middle fossa were encountered in more than half of the cases. A soft-tissue mass in the pterygoid fossa and posterior nasopharynx was recognized in 2 instances.

Pneumoencephalography and ventriculography are helpful in confirming the presence and location of the mass, but features pathognomonic of neurofibroma of the fifth cranial nerve were not found in this study.

Carotid angiography is helpful in excluding aneurysms and pituitary fossa tumors and in confirming the presence of a mass in the middle fossa.

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CEREBRAL ANGIOGRAPHY IN THE DIAGNOSIS OF SUPRASELLAR TUMORS*

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SUPRASELLAR tumors can be demonstrated by cerebral angiography as well as by pneumography. Pneumography is superior to angiography in that it gives an accurate indication of the size and shape of the tumor, as well as its relationship to the third ventricle, the foramen of Monro and the brainstem. Angiography is superior to pneumography in that tumor circulation can be visualized, thus making possible a specific histologic diagnosis, and in that an arterial aneurysm can be distinguished which by pneumography could be mistaken for a neoplasm. The latter is the primary reason why angiography has been growing in popularity in the study of all suprasellar syndromes—particularly in those cases where a meningioma is suspected or when an aneurysm cannot be excluded on clinical grounds. Also by carefully evaluating all of the findings on the angiogram, it is possible to arrive at a surprisingly accurate appreciation of the actual size of the tumor and its relationship to the sella turcica.

It is the purpose of this paper to point out the angiographic signs of suprasellar tumors and to establish criteria for determining size and direction of growth without resorting to pneumography.

PATHOLOGY

The tumors considered here are those which arise from the contents of the sella turcica, such as pituitary adenomas and craniopharyngiomas, or those which arise outside of the sella turcica. The latter group would comprise the suprasellar meningiomas, optic gliomas, epidermoid cysts (included with craniopharyngiomas by some) and aneurysms. Pituitary adenomas are by far the most common tumors in this region and were found in 43 of the 103 cases studied.

Pituitary adenomas may grow exclusively within the sella turcica by enlarging the osseous shell of the sella or they may break through the diaphragma sellae and project upwards. It is the tumor with suprasellar extension that presents with varying degrees of optic nerve and chiasmal compression. The extension of the tumor may be primarily upward but it may be upward and forward or upward and backward. It may also extend laterally.[2,5,11] A knowledge of the direction of growth as well as the extent of the tumor is desirable, particularly if surgery is contemplated.

The second group of tumors in order of frequency were the meningiomas. These may arise from the tuberculum sellae or the planum sphenoidale. They may not necessarily be attached to bone and may arise from the meninges around the cavernous sinus or around the optic nerves.

The third group of tumors studied were the craniopharyngiomas; some undoubtedly originated within the sella turcica and others arose apparently above the sella turcica. In this group are also included the suprasellar epidermoid cysts.

Aneurysms were also studied. These may produce signs of compression of the optic chiasm. In our experience those which caused optic nerve compression usually arose from the internal carotid artery just distal to or at the origin of the ophthalmic artery on the anterior or anteromedial aspect of the supraclinoid portion of the siphon.

Other lesions such as optic gliomas, metastatic pinealomas to the suprasellar region, nonneoplastic cysts and arteriovenous malformations represented but a

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small fraction of the cases analyzed.

Extradural tumors occurring in the region of the sella turcica, such as chordomas and nasopharyngeal carcinomas, will be considered in a subsequent publication.

MATERIAL AND CLASSIFICATION

This study consists of 103 patients, ranging in age from one to seventy-three years (Table I), with suprasellar tumors, who were examined by cerebral angiography. Sixty-two cases were proved by surgical exploration or postmortem examination, the remaining 41 by follow-up studies as well as clinical and roentgenologic evidence of the pathology (Table II). There are 44 males and 59 females in this group.

An exact pathologic diagnosis can be made by angiography only in aneurysms and in those few meningiomas which present a characteristic tumor circulation. Since the vast majority of tumors in our series are not in either of these groups, we have subdivided our cases according to the direction of growth rather than the histology (Fig. 1).

ANGIOGRAPHIC SIGNS OF SUPRASELLAR TUMORS

This problem has been considered by other authors.[6,7,8] The vessels of most significance in the evaluation of tumors in the region of the sella turcica are: the internal carotid artery; the proximal portion of the middle cerebral arteries; the horizontal and proximal ascending portions of the anterior cerebral arteries; the anterior choroidal artery; the posterior communicating and

Table I

AGE DISTRIBUTION OF 103 CASES WITH SUPRASELLAR AND PARASELLAR LESIONS

0–10	1
11–20	7
21–30	4
31–40	21
41–50	25
51–60	29
61–70	15
71 and over	1

Table II

DIAGNOSIS OF 89 CASES OF SUPRASELLAR AND PARASELLAR LESIONS

A. Pathologically Confirmed Diagnosis	
Pituitary adenomas	20
Meningiomas	14
Craniopharyngiomas	16
Aneurysms	5
Optic gliomas	3
Hypothalamic gliomas	1
Metastatic pinealomas	1
Arteriovenous malformation of interpeduncular space	1
Tortuous carotid arteries compressing optic chiasm	1
Total	62
B. Clinical and Roentgenologic Diagnosis Only	
Pituitary adenomas	23
Meningiomas	3
Aneurysms	1*
Total	27
Total Cases	89†

* One posterior suprasellar mass was considered on clinical and indirect roentgenographic evidence to be a basilar artery aneurysm. However, no vertebral angiography was performed.

† Fourteen cases with extradural tumors are not included.

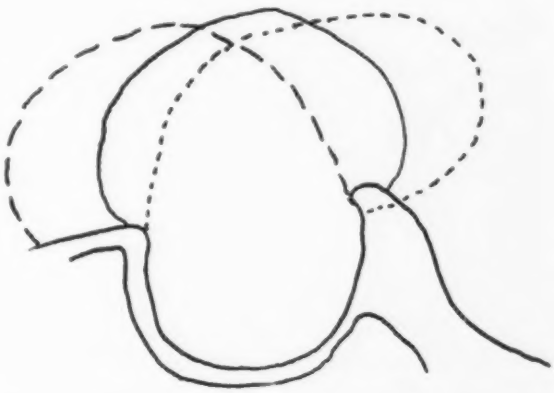

Fig. 1. Diagram of the characteristic directions of growth of suprasellar tumors. The solid line represents upward extension; the dashes represent anterior suprasellar extension; the dotted line represents posterior suprasellar extension. Lateral extension is not illustrated. It is apparent that an enormous tumor may occupy any or all of the above areas.

FIG. 2. Anteroposterior angiogram demonstrating the normal relationship between the anterior portion of the intracavernous segment of the internal carotid artery and the posterior portion (two arrows). The two points are superimposed. Normally the posterior portion is either superimposed upon or slightly medial to the anterior portion. The single arrow points to the anterior clinoid process which is about 0.5 cm. above the anterior end of the intracavernous segment. Note also the normal position of the posterior cerebral artery (p).

posterior cerebral arteries; and the basilar and internal cerebral veins.

I. CHANGES INVOLVING THE INTERNAL CAROTID ARTERY

In order to study the changes involving the internal carotid artery, the intracranial portion of this artery is divided into precavernous, intracavernous and supraclinoid segments.

The intracavernous segment may be displaced laterally. This has been observed pathologically[5,11] and demonstrated on base views of the skull during carotid angiography.[3] However, it is possible to demonstrate lateral displacement on the frontal angiograms as well. Normally, the posterior portion of the intracavernous segment is slightly more medial than the anterior portion (Fig. 2). This is easy to ascertain by following the artery as it comes out from the carotid canal of the petrous bone to determine the posterior portion; by tracing the supraclinoid segment downwards, it is possible to determine the anterior portion. It is sometimes difficult to ascertain whether lateral displacement is present or not, but if the posterior portion is lateral to the anterior portion, lateral displacement is probably present (Fig. 3).

Apparent upward displacement of the intracavernous segment is seen in pituitary adenomas and in craniopharyngiomas accompanied by sellar enlargement. This is due to depression of the floor of the sella turcica, the artery remaining in the same position (Fig. 4). Only tumors with extradural components cause absolute elevation of the intracavernous segment. A pituitary adenoma that extends epidurally may cause absolute upward displacement of this portion of the artery but this is uncommon.

The supraclinoid segment of the internal carotid artery may be: (a) displaced upward (opening of the siphon); (b) displaced downward and backward (closing of the siphon); (c) displaced laterally.

Upward displacement is characteristic of

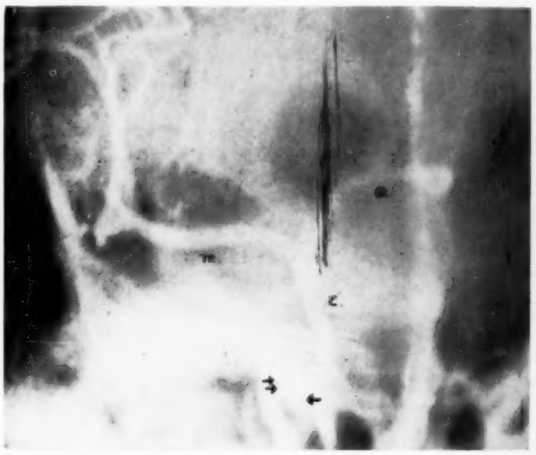

FIG. 3. A histologically verified pituitary adenoma in a fifty-four year old female (unit #137-02-85). The posterior portion of the intracavernous segment of the internal carotid (two arrows) is lateral to the anterior portion (one arrow). Note also the elevation of the carotid bifurcation with the stretching and lateral displacement of the carotid siphon (c) as well as the marked elevation of the anterior cerebral artery (a).

Fig. 4. Large pituitary adenoma (same case as in Figure 3). Dotted line outlines the apparent floor of the eroded sella turcica. Solid line represents the probable position of the floor prior to the erosion by the tumor. It can be seen that the intracavernous segment of the internal carotid artery is not elevated relative to the original floor of the sella turcica. The marked elevation of the carotid bifurcation (one arrow) and the stretching of the carotid siphon as well as the elevation of the proximal anterior portion of the cerebral artery (two arrows) are well demonstrated.

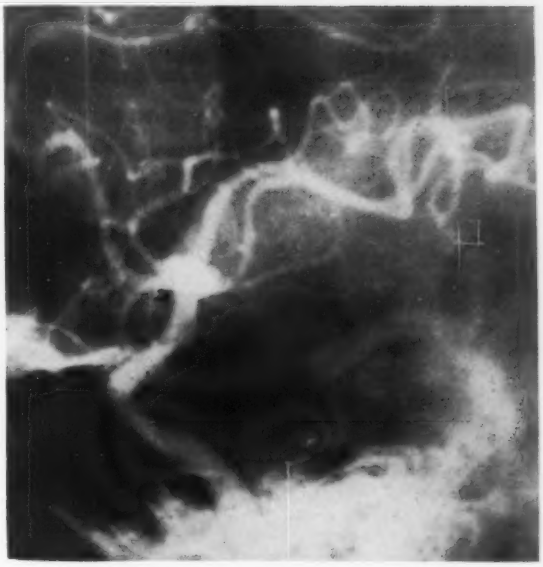

Fig. 5. Large pituitary adenoma in a fifty-two year old male (unit #30-00-70). There are marked elevation of the carotid bifurcation (arrow) and opening of the carotid siphon. The apparent elevation of the middle cerebral artery is related to the marked elevation of the bifurcation.

tumors with intrasellar origins such as pituitary adenomas (Fig. 5). Craniopharyngiomas do not displace the supraclinoid segment upward as frequently (Fig. 6, *A* and *B*), and may indicate in these cases that their origin is primarily extrasellar. Craniopharyngiomas may also originate within the sella turcica but they extend upward

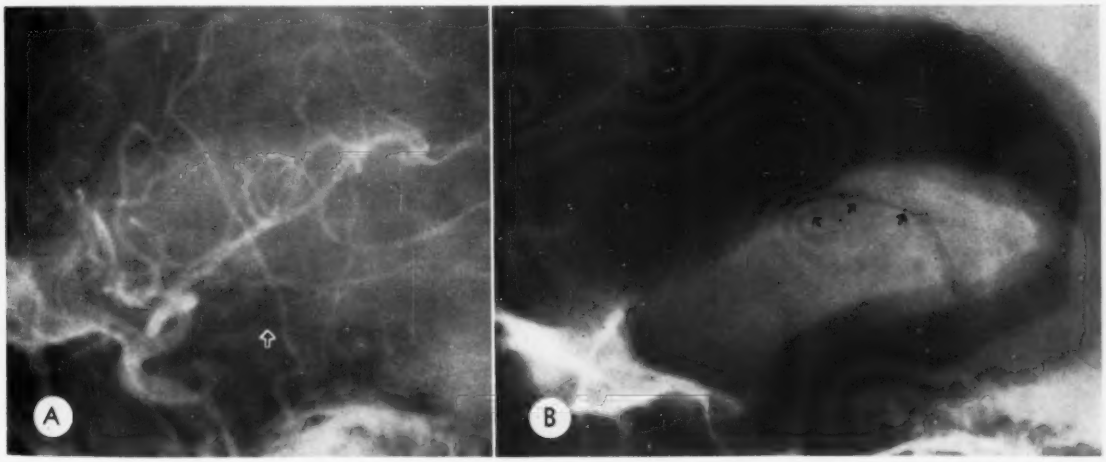

Fig. 6. Intraventricular craniopharyngioma in a thirty-two year old female (unit #133-01-22) who was admitted to the hospital with symptoms of chiasmal compression suggestive of a suprasellar mass. (*A*) The carotid siphon is normal in appearance. No vascular displacement is evident. The arrow points to a posterior branch of the middle meningeal artery not to be confused with a stretched anterior choroidal artery. (*B*) Ventriculogram showing obliteration of entire third ventricle by an enormous intraventricular craniopharyngioma. Arrows outline limits of tumor.

FIG. 7. Surgically verified craniopharyngioma in a seven year old female (unit #16-99-83). Thorotrast has been introduced into the suprasellar cyst (outlined by arrows). A narrow neck (two arrows) is seen connecting the large suprasellar portion with the small intrasellar portion. (Courtesy of Dr. John Scarff, New York City.)

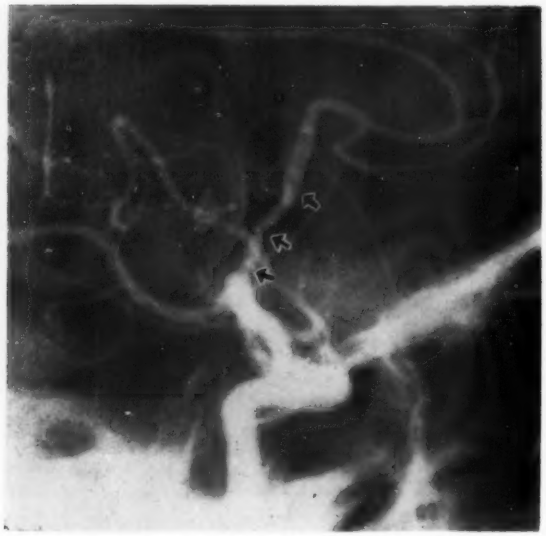

FIG. 8. Histologically proved anterior suprasellar craniopharyngioma in a forty-five year old male (unit #147-40-44). There are closing of the carotid siphon and marked elevation of the anterior cerebral artery (arrows).

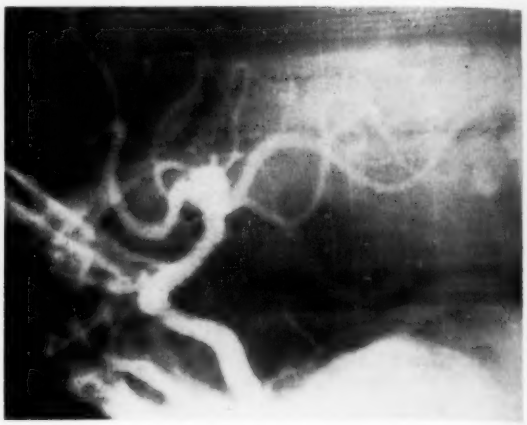

FIG. 9. Posterior suprasellar pituitary adenoma in a thirty-eight year old male (unit #133-03-05). The carotid siphon is open. The proximal portion of the anterior cerebral artery is not displaced upward. Parasellar (infratemporal) extension is also present (see Figures 11 and 17); the middle cerebral artery is elevated.

through the diaphragma sellae as a thin stalk which then mushrooms into a large cyst. In these cases one cannot expect elevation of the supraclinoid segment of the siphon (Fig. 7). This mode of extension is rare in pituitary adenomas.

Tumors in the anterior suprasellar region tend to produce downward and backward displacement of the siphon. This is also true in some pituitary adenomas with forward extension (Fig. 8).

Tumors in the posterior suprasellar region tend to open the siphon (Fig. 9); however, angiograms may show a normal appearance (Fig. 10*A*).

Lateral displacement of the supraclinoid segment of the internal carotid artery is almost always present when tumors lie medially to the vessel. This is true in most cases of pituitary adenomas as well as in most of the other suprasellar tumors (Fig. 3). Medial displacement may be encountered in cases of pituitary adenomas that have extended laterally (Fig. 11). Stretching of the supraclinoid segment and elevation of the bifurcation of the internal carotid artery are fairly common findings in suprasellar tumors, in general.

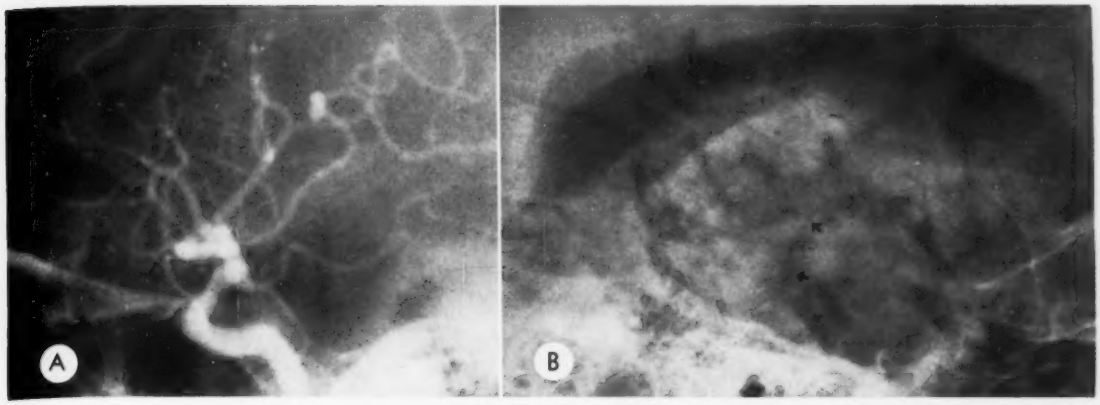

Fig. 10. Posterior suprasellar pituitary adenoma in a sixty year old female. (*A*) Lateral arteriogram shows normal siphon and no definite vascular displacements. (*B*) Lateral pneumoencephalogram demonstrates the extensive tumor mass (arrows).

II. CHANGES INVOLVING THE ANTERIOR CEREBRAL ARTERY

The horizontal portion of the anterior cerebral artery is usually displaced upward in tumors that are either directly suprasellar or anterior suprasellar in location (Fig. 3 and 4). In posterior suprasellar tumors the anterior cerebral artery will not be affected even by large tumors (Fig. 6, *A* and *B*; 9; 10, *A* and *B*; and 11). It is often extremely difficult to determine whether the horizontal portion of the anterior cerebral artery is or is not displaced upward, particularly in view of the fact that some degree of elevation may be found in normal individuals and even greater apparent elevation may be seen in individuals with vascular tortuosity (Fig. 12). This apparent elevation is frequently seen in children.[10] The degree of elevation of the anterior cerebral artery is usually determined on the frontal angiograms but roentgenograms with increased angulation showing the orbits may be needed as well as the straight anteroposterior ones. A more reliable sign may be seen on the lateral angiogram (Fig. 4 and 8). Normally, the curve is convex downwards, immediately distal to the siphon. In the presence of anterior suprasellar tumor, this convexity may be flattened or a concavity may be present (Fig. 4, 8 and 13). Posterior displacement of the anterior cerebral artery may occur in the presence of large anterior suprasellar tumors and is usually associated with upward displacement of the vessel.

III. CHANGES IN THE MIDDLE CEREBRAL ARTERIES

Suprasellar tumors may elevate the proximal portion of the middle cerebral arteries;

Fig. 11. Large posterior suprasellar pituitary adenoma with lateral extension (same case as in Figure 9). The elevation of the middle cerebral artery (arrow) is greater than the elevation of the carotid bifurcation. The carotid siphon is stretched and medially displaced. No elevation of the anterior cerebral artery is present. (Compare with Figure 3.)

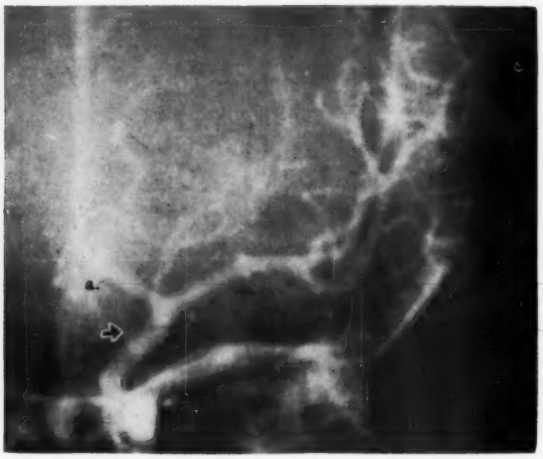

FIG. 12. Patient with no clinical evidence of chiasmal compression or endocrine changes. There is apparent elevation of the horizontal portion of the anterior cerebral artery (a). This is a frequent normal finding (see text). The posterior communicating artery is in a normal position (arrow).

this elevation is usually an indication of the upward displacement of the carotid bifurcation and only occasionally is it due to infratemporal extension of suprasellar tumors (Fig. 3 and 11). Lateral displacement of the ascending branches of the middle cerebral arteries may be found in cases of massive suprasellar tumors which produce ventricular dilatation secondary to obstruction of the foramen of Monro.

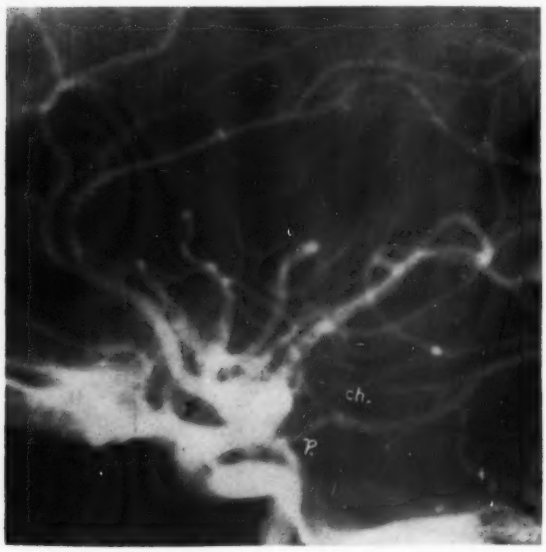

FIG. 13. Normal lateral arteriogram demonstrating the downward convexity usually seen in the proximal portion of the anterior cerebral artery (arrow). Compare with Figures 4, 8 and 17. The anterior choroidal (ch) and posterior communicating (p) arteries are in normal positions.

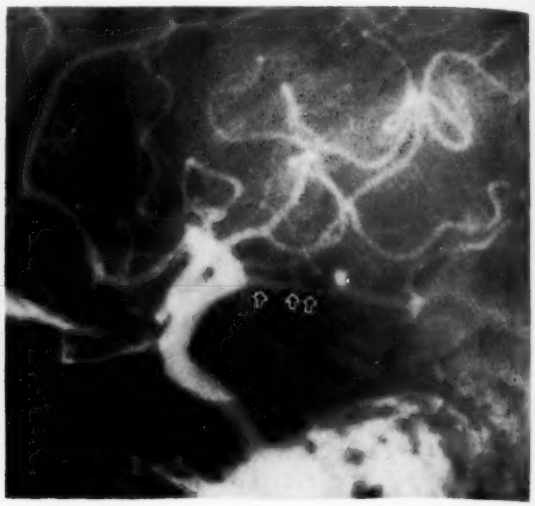

FIG. 14. A histologically verified pituitary adenoma in a sixty-two year old female (unit #24-98-13). There are elevation and forward displacement of the carotid bifurcation and elevation of the posterior communicating and anterior portion of the posterior cerebral arteries (arrows).

IV. CHANGES IN THE POSTERIOR COMMUNICATING AND POSTERIOR CEREBRAL ARTERIES

The posterior communicating and posterior cerebral arteries may be deflected superiorly and laterally by posterior suprasellar tumors (Fig. 14 and 15, *A*, *B* and *C*). Upward displacement of the posterior communicating and proximal posterior cerebral arteries may be very difficult to evaluate. Lateral displacement, as seen on the frontal angiogram, is easier to ascertain (Fig. 15*A*). Normally, the posterior communicating artery extends posteriorly and medially from its origin in the internal carotid artery. When laterally displaced, the posterior portion is either superimposed upon or lateral to its anterior portion. The presence of a posterior suprasellar mass can often be determined on angiograms only by the lateral displacement of the posterior communicating, posterior cerebral and anterior choroidal arteries. If these arteries do not

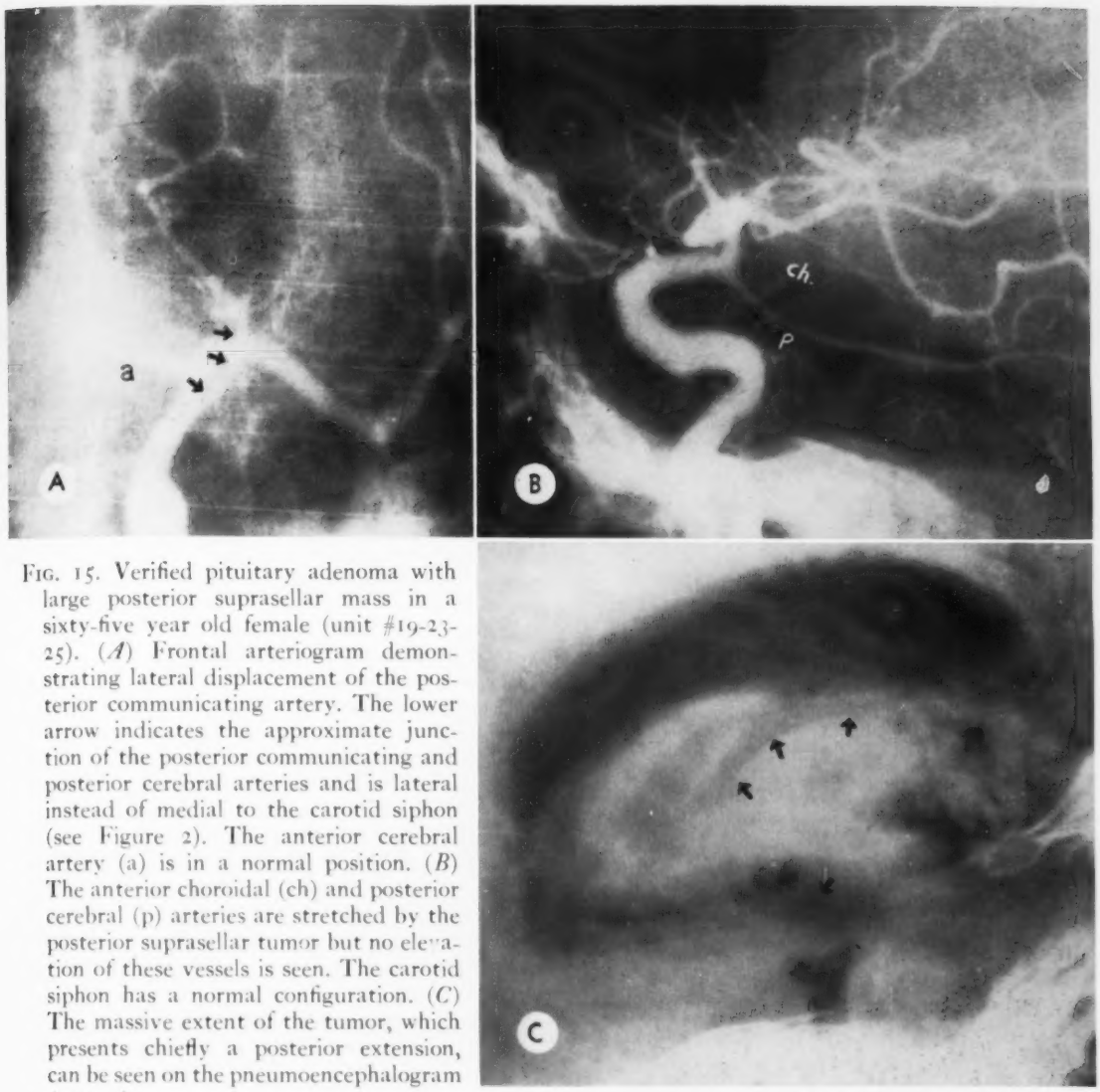

Fig. 15. Verified pituitary adenoma with large posterior suprasellar mass in a sixty-five year old female (unit #19-23-25). (*A*) Frontal arteriogram demonstrating lateral displacement of the posterior communicating artery. The lower arrow indicates the approximate junction of the posterior communicating and posterior cerebral arteries and is lateral instead of medial to the carotid siphon (see Figure 2). The anterior cerebral artery (a) is in a normal position. (*B*) The anterior choroidal (ch) and posterior cerebral (p) arteries are stretched by the posterior suprasellar tumor but no elevation of these vessels is seen. The carotid siphon has a normal configuration. (*C*) The massive extent of the tumor, which presents chiefly a posterior extension, can be seen on the pneumoencephalogram (arrows).

fill well, the tumor may be overlooked (Fig 6, *A* and *B*; and 10, *A* and *B*).

V. Changes in the Anterior Choroidal Artery

The origin of the anterior choroidal artery is in the internal carotid artery, 1 or 2 mm. distal to the origin of the posterior communicating artery. As was true with the posterior communicating and posterior cerebral arteries, posteriorly placed suprasellar masses may cause upward and lateral displacement of the anterior choroidal arteries. Upward displacement is often very difficult to evaluate unless it is massive. Lateral displacement may be determined on the frontal angiograms. This is usually present when the most medial portion of the artery (the portion that passes about the uncus) is superimposed on or lateral to the internal carotid artery. Normally, this segment projects medially to the siphon (Fig. 16 and 17). Inferior and lateral displacement of the anterior choroidal and posterior communicating arteries may occur with posterior suprasellar tumors that

FIG. 16. Posterior suprasellar craniopharyngioma in a fifty-seven year old male (unit #148-36-40). There is considerable lateral displacement of the anterior choroidal artery (arrows).

are extrasellar in origin, particularly in cases of tumors originating in the third ventricle.

VI. CHANGES IN THE INTERNAL CEREBRAL VEIN AND THE BASILAR VEIN

The internal cerebral vein and the basilar vein are seldom noted to be affected by suprasellar tumors. Tumors which extend laterally may cause elevation of the basilar vein (Fig. 18). There were 8 such cases in our series. Massive suprasellar tumors may elevate the anterior portion of the internal cerebral vein (Fig. 19).

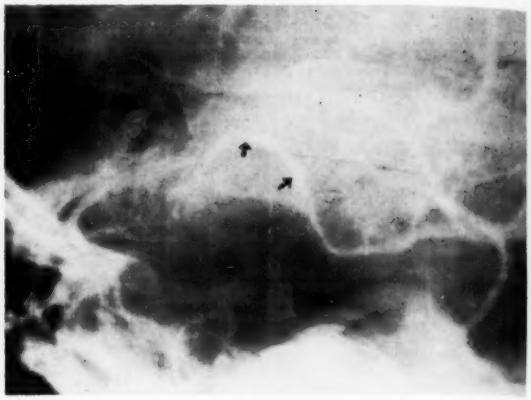

FIG. 18. Same case as in Figures 9 and 11. Marked elevation of the basilar vein can be seen with infratemporal extension of a pituitary adenoma (arrows).

COMMENT

In addition to aneurysms, pituitary adenomas can be diagnosed accurately by angiography. It is apparent that all pituitary adenomas begin as intrasellar tumors. The degree of erosion and expansion of the sella turcica depends primarily on the strength and completeness of the diaphragma sellae. Since the medial dural wall of the cavernous sinus is relatively thin and flexible, an expanding process in the sella

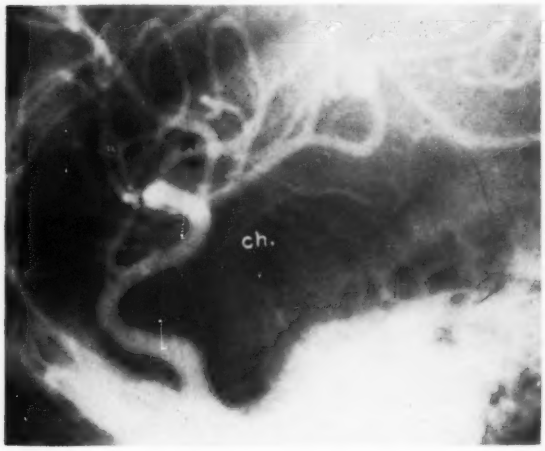

FIG. 17. A histologically verified large suprasellar pituitary adenoma in a forty year old male (unit #20-73-07). There is elevation of the carotid siphon as well as elevation of the anterior cerebral artery (arrows) and anterior choroidal artery (ch).

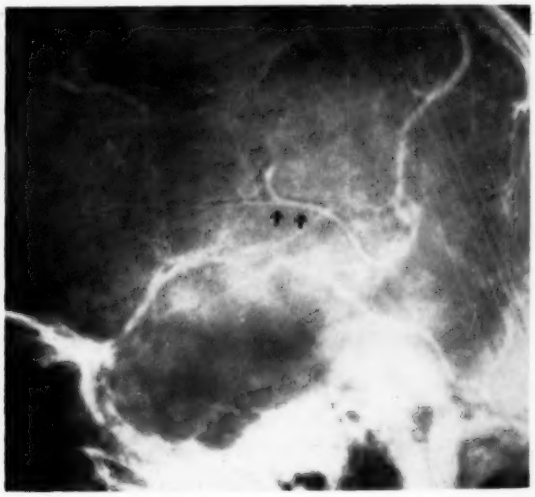

FIG. 19. Enormous pituitary adenoma in a fifty-one year old male (unit #29-98-89) causing elevation of the internal cerebral vein (arrows).

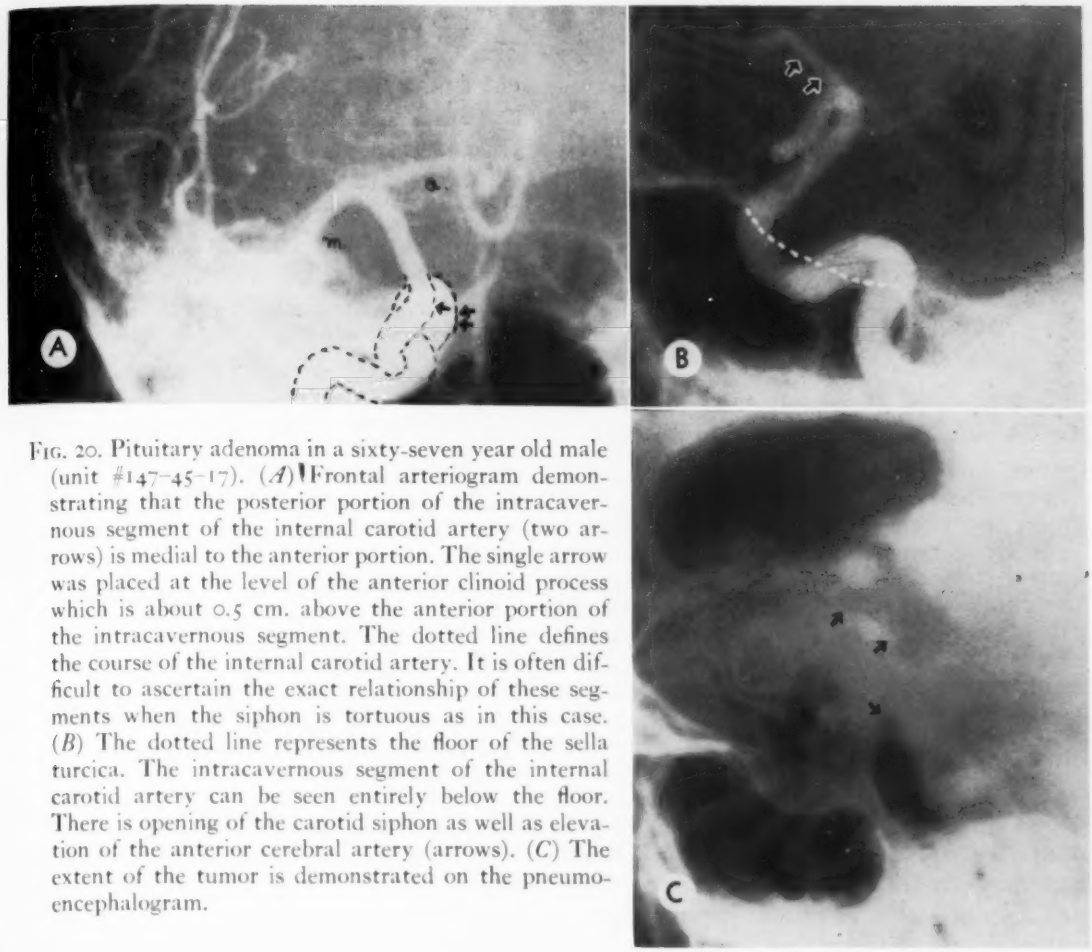

Fig. 20. Pituitary adenoma in a sixty-seven year old male (unit #147-45-17). (*A*) Frontal arteriogram demonstrating that the posterior portion of the intracavernous segment of the internal carotid artery (two arrows) is medial to the anterior portion. The single arrow was placed at the level of the anterior clinoid process which is about 0.5 cm. above the anterior portion of the intracavernous segment. The dotted line defines the course of the internal carotid artery. It is often difficult to ascertain the exact relationship of these segments when the siphon is tortuous as in this case. (*B*) The dotted line represents the floor of the sella turcica. The intracavernous segment of the internal carotid artery can be seen entirely below the floor. There is opening of the carotid siphon as well as elevation of the anterior cerebral artery (arrows). (*C*) The extent of the tumor is demonstrated on the pneumoencephalogram.

turcica will usually affect the cavernous sinus, displacing it laterally and expanding the floor.[5,11] This can be demonstrated by angiography except in those cases in which the sella turcica is shallow and associated with extensive pneumatization of the sphenoid sinus (Fig. 20, *A*, *B* and *C*). Individuals with this anatomic variant often have an incomplete or a weak diaphragma sellae. Also in these instances, most of the cavernous sinus lies below the level of the floor of the sella turcica which is usually normal in size. Because the weak diaphragma sellae offers little resistance to the upward expansion of the tumor, lateral displacement of the intracavernous portion of the internal carotid artery does not occur.[4] There were 4 cases of this type in this series.

Small tumors of the tuberculum sellae are difficult to detect angiographically since the anterior cerebral arteries, the only vessels that might be displaced by these tumors, are not affected. One case of a small meningioma arising from the optic nerve sheath showed no angiographic abnormalities. Indeed, neither are direct changes seen on pneumoencephalograms with the exception of the distortion of the parolfactory sulci.

It should be mentioned that some craniopharyngiomas or "suprasellar gliomas" may arise in the hypothalamus, and in these cases no specific vascular displacement may be seen other than lateral displacement and occasionally downward displacement of the anterior choroidal and/or

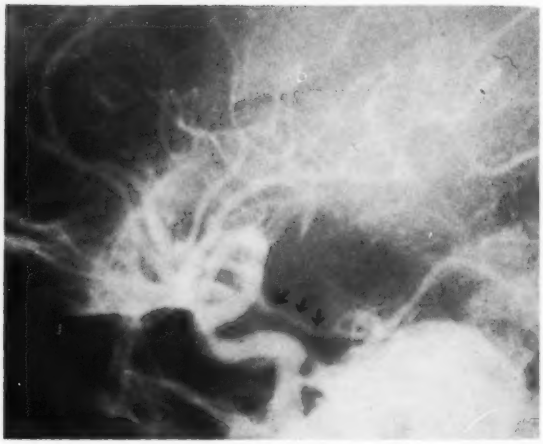

FIG. 21. Intraventricular craniopharyngioma in a forty year old female (unit #21-08-25). The arteriogram demonstrates downward displacement of the posterior communicating and posterior cerebral artery (arrows). Ventriculograms showed marked ventricular dilatation and a large mass filling the third ventricle almost completely. At operation the tumor proved to be an intraventricular craniopharyngioma.

posterior communicating arteries (Fig. 21).

Posterior suprasellar tumors with no intrasellar component were the most difficult to diagnose by angiography. These tumors, even though relatively large, may not displace the anterior choroidal, the posterior communicating and posterior cerebral arteries; and even if these vessels are visualized, the tumors will escape detection (Fig. 6, *A* and *B*).

In 54 of the 103 cases reviewed, pneumography as well as angiography was performed. Pneumography was felt to be superior in 48 per cent of these cases; both procedures were considered equal in 27 per cent, and arteriography was considered superior in 25 per cent.

SUMMARY

One hundred and three cases of suprasellar tumors with angiographic studies have been reviewed. One-half of these had pneumography as well. In two-thirds of the cases the diagnosis was confirmed at surgery or autopsy. In the majority of cases the diagnosis can be made by angiography alone.

The vessels of most significance in the evaluation of these tumors are the precavernous, intracavernous and supraclinoid segments of the internal carotid arteries; the anterior choroidal arteries; the posterior communicating and posterior cerebral arteries; and the proximal portion of the middle cerebral and the anterior cerebral arteries. The basilar and internal cerebral veins have occasionally been of value.

In most instances angiography has equaled pneumography in establishing the diagnosis of suprasellar tumors. In the majority of cases of pituitary adenomas, angiography has been superior to pneumography in determining the type of tumor. The lateral displacement of the intracavernous portion of the internal carotid artery is fairly characteristic of pituitary adenoma as against other types of suprasellar tumors not originating within the sella turcica. *Anterior suprasellar* masses may be distinguished by the elevation of the anterior cerebral arteries and the posterior displacement of the supraclinoid segment of the internal carotid arteries (closing of the siphon). *True suprasellar* tumors usually produce elevation and lateral displacement of the carotid siphon, elevation of the carotid bifurcation (opening of the carotid siphon), and, occasionally, lateral displacement of the anterior choroidal and/or posterior communicating arteries. *Posterior suprasellar* tumors are the most difficult to diagnose angiographically. When diagnosable, they have caused lateral displacement and elevation of the anterior choroidal and posterior communicating and posterior cerebral arteries. It has not been possible in most instances to determine the posterior extent of these tumors by angiography alone. Tumors which extend laterally may lift the temporal lobe and thus produce elevation and lateral displacement of the middle cerebral artery and basilar vein. They may also cause medial displacement and elevation of the supraclinoid segment of the internal carotid artery.

Aneurysms presenting with chiasmal syndromes have been found to originate

primarily from the internal carotid artery at or near the junction of the ophthalmic artery and may not be differentiated by pneumography from tuberculum sellae or planum sphenoidale tumors. It is usually necessary to perform bilateral angiography when studying suprasellar tumors in order to rule out an aneurysm, for there may be nonfilling of the aneurysm from one side even on cross compression.

Bilateral angiography is also of value for a comparison of both sides in determining the presence of small tumors causing relatively subtle changes in the vessels of the circle of Willis.

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COMPARISON OF CEREBRAL ANGIOGRAPHIC CONTRAST MEDIA BY THEIR CIRCULATORY EFFECTS*

AN EXPERIMENTAL STUDY

By HARRY W. FISCHER, M.D.,† *and* JOHN W. ECKSTEIN, M.D.‡

CEREBRAL angiography has never been a completely innocuous procedure. Despite improvements in techniques and in contrast materials, a significant incidence of complications yet remains. These complications are transient and mild, or permanent and severe. To know whether the complications following cerebral angiography are due to the contrast material and the injection technique or to the progression of the investigated disease process is difficult. Most intracranial lesions in which cerebral angiography is indicated are capable of exhibiting sudden worsening of symptoms and signs at any time in their course. Adequate clinical comparison of contrast media requires large groups of patients with identical lesions and clinical courses, so that differences in postangiographic status can be correctly ascribed to the contrast material. Although clinical studies may be planned with great care, the lack of sufficient numbers of comparable patients and the great unpredictable variability of disease processes do not allow proper experimental control.

Animal experimentation, therefore, occupies an important place in the evaluation of the contrast media in cerebral angiography. We should say that any satisfactory contrast material must meet two requirements. It should be of a physicochemical nature to supply the information desired, and it should disturb the normal working of the body little or not at all.

We have concerned ourselves with the second requirement, the effect of the contrast materials on the body, but the first requirement, which is largely based on radiopacity, is related to our problem.

Evaluation of cerebral angiographic contrast agents by the degree of response of organs, or the body as a whole, has been obtained in several ways (Fig. 1). The immediate and residual neurologic status of the subject can be observed and expressions of distress, involuntary movements, the severity and type of convulsions measured. Electroencephalography, electrocardiography, recording of blood pressures, cerebral spinal fluid pressure and cerebral blood flow and respiratory measurements give more quantitative and delicate information on the less apparent effects of cerebral angiography. Testing of altered permeability of the blood-brain-barrier and histologic studies of the organs have revealed cytotoxic effects.

We selected the alterations in arterial blood pressure, venous pressure, heart rate and rhythm, the electrocardiogram and end-expiratory CO_2 concentration resulting from experimental cerebral angiography as refined, sensitive indications of the toxicity of a particular contrast material. Alterations of this type in part were first noted by Weatherall,[26] and later by Broman and Olsson,[4] Foltz *et al.*[9] Bassett and Rogers,[1] Schmidt,[22] and Lindner *et al.*[19] Greitz[10] studied the same type of phenomena in man, and Lindgren and Törnell[16,17] recently made more detailed studies in cats. Several reasons prompted the preference of this to previous methods. The present study allowed an evaluation based on amounts and concentrations of material comparable

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to those used in clinical cerebral angiography, whereas most of the previous studies were based upon injection of amounts so excessive as to be unrelated to clinical usage. The measured vital signs are of well understood importance to normal functioning. Any disturbance of these signs by a diagnostic roentgenologic procedure is therefore worthy of consideration. The present method of evaluation was performed on the intact animal in a fashion very similar to clinical angiography, and can be applied to the human patient without difficulty. The measurement of change is quantitative, graphic and nonsubjective and does not require death of the animal.

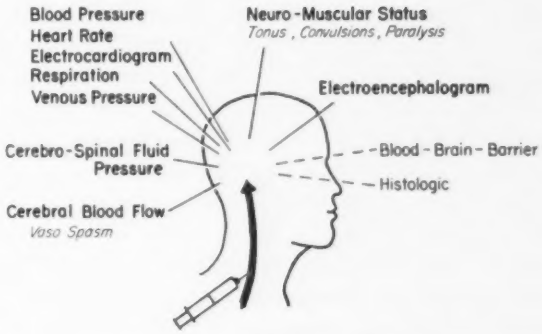


Fig. 1. Contrast media for cerebral angiography have been compared experimentally for their toxic effects by one or more of the above illustrated methods of detection.

DESIGN OF EXPERIMENTS

The injections were given in two separate patterns:

Pattern I. Originally, two contrast materials were compared in a single dog by giving a series of 5 or 6 injections of which injections 1, 3 and 5 were of contrast material A, and injections 2, 4 and 6 were of contrast material B. Equal amounts of the two contrast materials were compared. The doses injected were 0.14 to 0.42 cc. per kg. of body weight of 50 per cent solutions of hypaque, urokon, diodrast, miokon and cardiografin. Only one dose level was tested per dog. Dissatisfaction with this design resulted because of the wide range of response of individual dogs, and because we reasoned that the contrast agents, although of equal chemical concentration and approximately equal tonicity, were not being compared on an equal basis as to radiopacity.

Pattern II. Three contrast materials, hypaque, urokon, and cardiografin, were compared in each of 6 dogs. Two dose levels were tested, 0.15 and 0.30 cc. per kg. of body weight. A Latin square[8] was used to eliminate variability among dogs by randomization of the order of injection. Two doses were selected in which a measurable response was anticipated. Letters A and B of the square were assigned to 0.15 cc. and 0.30 cc./kg. amounts of 50 per cent hypaque, C and D to like amounts of 61 per cent cardiografin, and E and F to like amounts of 45.5 per cent urokon. Hypaque was chosen as a standard because of its wide clinical use. Urokon and cardiografin concentrations were adjusted to equate their iodine contents to that of 50 per cent hypaque.

CONDITIONS PERTINENT TO BOTH PATTERNS OF INJECTION

All experiments were performed on adult dogs, 10 and 20 kg., under intravenous nembutal solution sufficient to induce and maintain surgical anesthesia. Respiration was maintained at a constant rate and volume, beginning immediately after anesthetization by a Harvard fixed volume respirator-pump connected to an inlying intratracheal tube with inflated cuff. Respiration of constant rate and volume is not normal for clinical angiography, but we thought it appropriate to eliminate variability in respiration. Other responses could then be better analyzed. With the dog in the recumbent position, the right common carotid artery was exposed in the lower neck. Just prior to each puncture and injection the carotid was proximally clamped, to minimize inconstancy of mixing of the cerebrally flowing contrast material by blood. All injections were made by the same operator individually puncturing the artery with a 20-gauge needle. Injections

were from a 10 cc. syringe, by manual pressure in Pattern I trials, and by a Shipps injector in Pattern II. The use of the pressure injector required an interconnecting polyethylene tube between needle and syringe. The Shipps injector allowed equating injection times of contrast solutions of different viscosity. Successive injections were made at 15 to 25 minute intervals to allow return of conditions to a pre-injection level insofar as possible.

A Cournand needle was placed in the right femoral artery. A cardiac catheter was inserted into the vena cava through the right femoral vein. Mean arterial blood pressure was determined by electrical integration of the Statham strain gauge output and the pressure response was expressed in mm. Hg. The venous pressure, also measured by a Statham strain gauge, was expressed as the peak response in mm. Hg.

Electrocardiograms were obtained, using the standard leads. The heart rate reduction was expressed in beats per minute for an arbitrary ten second period after injection. This length of time was sufficient to include the major periods of asystole, pronounced bradycardia and arrhythmia. A Beckman medical gas analyzer (Spinco Model LB-1) was connected to the intratracheal tube for the CO_2 measurements. Ventilation was adjusted so that the animal was well oxygenated, with his end-expiratory CO_2 concentration about 4 per cent. Changes in CO_2 concentration were then expressed in arbitrary units. The Sanborn direct writing oscillograph allowed simultaneous tracings of all measurements.

In the Latin square experiment, the responses to each dose level of each contrast solution were averaged. When individual responses were expressed as decrease in mm. Hg or decrease in heart rate, *et cetera*, the baseline level was approximately the same before each response. Responses were expressed as decreases in mm. Hg or beats per minute from baseline, not as percentage decrease.

The contrast materials were taken from ordinary commercial vials, not previously exposed to air or light. Bubbles and small clots were avoided.

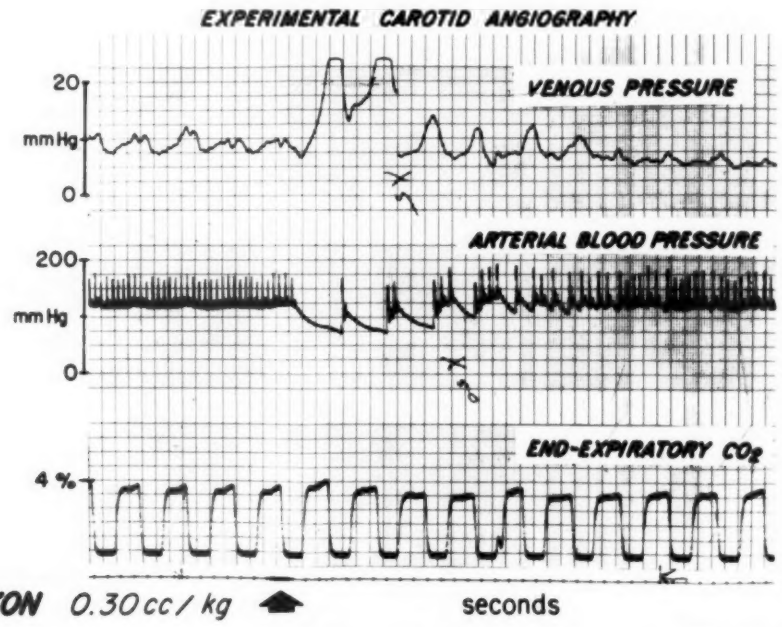


FIG. 2. A typical recording of the cardiovascular disturbances following the intracarotid injection of a contrast solution in the dog, in this case 0.30 cc./kg. of 45.5 per cent urokon. The periods of asystole, bradycardia, arterial hypotension, elevation of venous pressure, and decrease in end-expiratory CO_2 content are to be noted. The period of injection is indicated by the black bar on the time line.

Table I

		Cardiografin	Hypaque	Urokon
Arterial Blood Pressure Fall (mm. Hg)				
0.15 cc./kg.	Range	0–20.0	0–60.0	5.0–130.0
	Average	5.0	18.3	53.0
0.30 cc./kg.	Range	5.0–75.0	5.0–95.0	60.0–120.0
	Average	26.7	56.0	91.0
Duration of Major Blood Pressure Abnormality (seconds)				
0.15 cc./kg.	Range	0–14.0	0–18.0	2.0–31.0
	Average	3.3	5.3	10.8
0.30 cc./kg.	Range	0–15.0	2.0–20.0	2.0–21.0
	Average	5.0	8.0	12.5
Decrease in Heart Rate in the 10 Seconds Post Injection (beats per minute)				
0.15 cc./kg.	Range	0–78.0	6.0–132.0	18.0–144.0
	Average	16.8	36.0	78.0
0.30 cc./kg.	Range	12.0–72.0	24.0–156.0	48.0–192.0
	Average	34.0	66.0	125.0
Peak Rise in Venous Pressure (mm. Hg)				
0.15 cc./kg.	Range	0–1.5	0.5–3.5	0– 5.0
	Average	0.7	1.3	3.0
0.30 cc./kg.	Range	0.5–4.0	0.5–4.0	2.5–11.0
	Average	1.4	3.1	6.3
Reduction in End-Expiratory CO_2 Concentration (arbitrary units)				
0.15 cc./kg.	Range	0.5–2.0	0.5–3.0	1.0–7.0
	Average	1.0	1.5	5.0
0.30 cc./kg.	Range	0.0–5.0	1.0–9.0	3.0–9.0
	Average	1.5	3.7	6.2

RESULTS

The most complete information was obtained from the Pattern II injections, comparing the animals' responses to cardiografin, hypaque, and urokon of approximately equal iodine content. Similar, but on the whole less precise, information was obtained from the Pattern I injections, in which 50 per cent solutions of cardiografin, diodrast, hypaque, miokon and urokon were injected.

The responses depended upon the contrast material and its dose. In almost every instance a greater disturbance was produced by the dose of 0.3 cc./kg. than by the 0.15 cc./kg. dose of each of the three contrast agents.

A typical positive response consisted of pronounced slowing of the heart and/or a brief period of asystole. The response began before the completion of the injection or within two seconds after completion. The electrocardiographic changes were those usually associated with vagal stimulation of the heart. Pronounced bradycardia and short periods of asystole were the most common forms of arrhythmia. Atrial and ventricular premature beats were seen rarely. Other focal arrhythmias were not seen, nor were there instances of atrial or ventricular fibrillation. With slowing or standstill of the heart, a fall of arterial blood pressure occurred, the magnitude of which correlated with the severity and duration of the changes in heart rate. Occurring with the bradycardia, asystoles, and hypotension was an elevation of venous pressure. End-expiratory CO_2 concentration decreased simultaneously. An explanation of the end-expiratory CO_2 concentra-

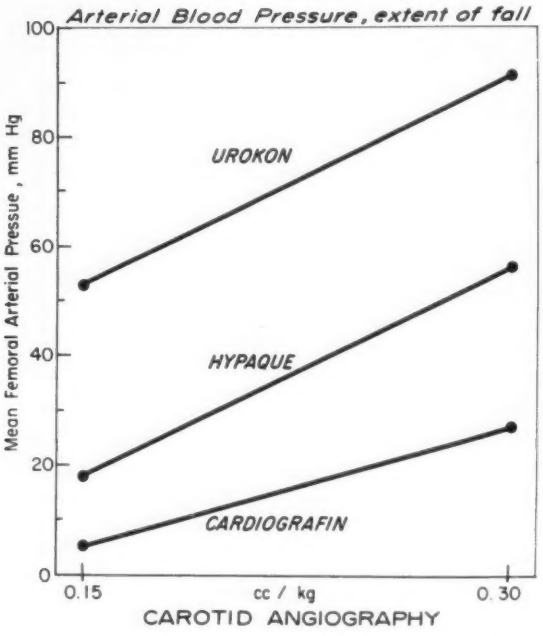


FIG. 3. Graph showing a comparison of the dose-response curves of the three contrast media at 0.15 cc./kg. and 0.30 cc./kg. in regard to extent of fall of arterial blood pressure. (Each point on Figure 3 through 7 is an average of the response of 6 dogs).

tion measurements is as follows: An acute change in end-expiratory CO_2 concentration at a fixed rate of ventilation is a function of the flow of blood through the lungs. This is equal to the cardiac output. A decrease in CO_2 concentration occurring before any change in respiratory quotient could become manifest must be related to a reduction in pulmonary perfusion.[21] In these experiments the acute reduction in CO_2 concentration is interpreted as a decrease in cardiac ouput. This reduction of output in the presence of an increased venous pressure, which is a function of filling pressure, is evidence of an acute reduction in the functional state of the myocardium. This might be secondary to vagal stimulation which is known to reduce myocardial performance directly as well as to reduce output by cardiac slowing.[3] The record of a typical response is shown in Figure 2.

The decisive differences in responses are presented in Table 1 for Pattern II injections.

Summarizing the Pattern II injections, cardiografin caused the least disturbance in the measured cardiovascular functions. Hypaque was intermediate. Urokon produced the most constant and severe disturbances. Although no 2 animals responded to the same degree to a given injection, the relative responses were consistent. In 30 comparisons (of 60 injections) between hypaque and cardiografin, the disturbances caused by hypaque were clearly greater in 14 instances, equal in 13, and less in only 3. In 30 comparisons between urokon and cardiografin, the differences were even greater. Urokon caused more disturbance in 28 instances, and in only 2 instances were the responses equal. At no time did cardiografin cause more disturbance than urokon. Hypaque caused greater disturbances than urokon on only 3 occasions. In 7 comparisons, the responses to hypaque and urokon were equal, and in the other 20 comparisons urokon caused more disturbance than hypaque. The differences in responses to the three contrast media and the dose-response curves for each medium are shown

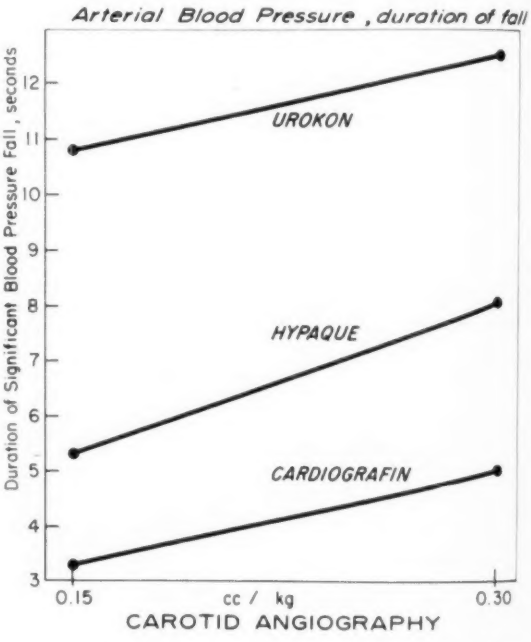


FIG. 4. Graph showing a comparison of the dose-response curves of the three contrast media at 0.15 cc./kg. and 0.30 cc./kg. in regard to duration of fall of arterial blood pressure.

in Figures 3 to 7. Comparison of blood pressure and heart rate for the three media at a dose of 0.15 cc./kg. are shown in 2 dogs. The responses of animal No. 289 in Figure 8 were greater than those of dog No. 290 in Figure 9. The relative responses to the three media were the same.

Of the first group of experiments (Pattern 1 injections), the types of responses were the same. The number of dogs injected and the variation of response of individual dogs do not allow more precise tabulation of data, but it was clearly seen that cardiografin was consistently least disturbing to heart rate and rhythm, the electrocardiogram, and arterial and venous blood pressure. Urokon was most consistently and severely disturbing. No overlap in the degree of response was noted between urokon and cardiografin. The responses to diodrast, hypaque and miokon were more or less of the same degree and were intermediate in magnitude between cardiografin and urokon. The responses of the three could not be distinguished consistently from each other. With these same three compounds, some overlap in response with urokon was noted at one end of the scale. At the other, less reactile end of the scale, some but less overlap in degree of response with cardiografin was observed.

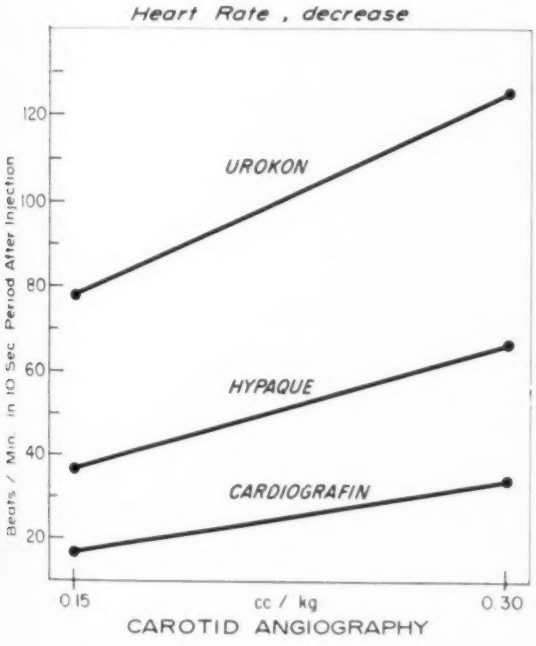


Fig. 5. Graph showing a comparison of the dose-response curves of the three contrast media at 0.15 cc./kg. and 0.30 cc./kg. in regard to decrease in heart rate.

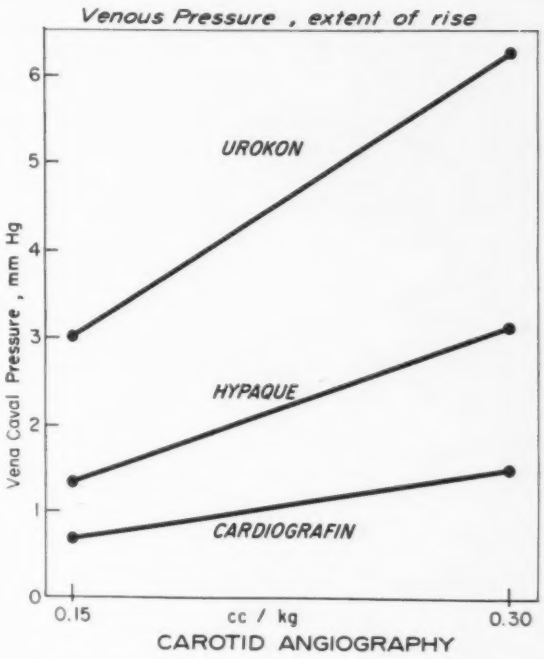


Fig. 6. Graph showing a comparison of the dose-response curves of the three contrast media at 0.15 cc./kg. and 0.30 cc./kg. in regard to the extent of rise of venous pressure.

COMMENT

The value of these experiments is based upon two points. First, cerebral angiographic contrast materials vary widely with respect to their pharmacologic side effects. Secondly, the observed disturbances in blood pressure, heart rate and rhythm, and cardiac output could be of clinical significance.

Toxicity of a cerebral angiographic agent is measured by disturbance in a body function or alteration of a body structure during or following its injection. Slowing of the heart and disturbance of its rhythm, lowering of arterial pressure, raising of venous pressure, and decreasing of cardiac output result from an injection of a fraction of the dose required to produce electoencephalographic abnormalities, increase in blood-brain barrier permeability or damage to the

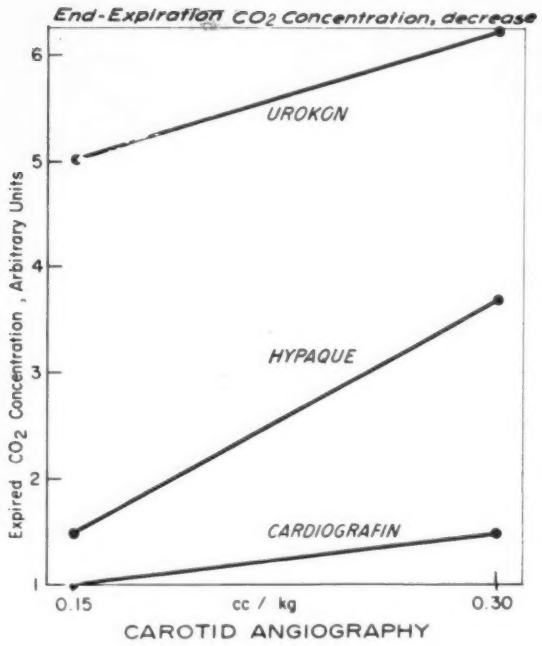


FIG. 7. Graph showing a comparison of the dose-response curves of the three contrast media at 0.15 cc./kg. and 0.30 cc./kg. in regard to decrease of end-expiration CO_2 concentration.

cord by experimental lumbar aortography. The amounts in the present study were 0.14 to 0.42 cc. per kg. of body weight, injected as single doses. A study of toxicity based upon change in blood-brain-barrier permeability utilized single doses of 0.7 to 3.7 cc./kg., an amount estimated to be 10 times the single injection dose in man.[27] Another blood-brain-barrier study was based upon injection into the rabbits' carotid artery of amounts 375 times as great as would be used for man.[28] Electroencephalography is not a very sensitive measure of toxicity. At least, 1.3 cc./kg. of 50 per cent diodrast was required to produce electroencephalographic abnormalities in rabbits.[2] Higher doses were more consistent in producing changes. Ingvar and Söderberg[12] found no changes in the electroencephalogram with doses comparable to those used clinically.

Experimental lumbar aortography, which has been useful in comparing toxicity of angiographic contrast agents, was based essentially on doses of 0.5 to 3.0 cc./kg. of 70 per cent concentration.[25] In clinical cerebral angiography a single injection in an adult human varies from 0.06 to 0.2 cc. per kg. of body weight, depending upon the selection of a 6 or 10 cc. injection and the body weight of the patient. Although the gross dose for the dog in the present experiments is greater than for man, consideration of the dog's carotid circulation minimizes the difference. In the dog, blood supply to the brain is derived from an internal carotid artery having a diameter a fraction of the size of the common or external carotid. The internal carotid receives a minor portion, the external carotid a major portion of the common carotid's blood flow. In man, the internal carotid artery receives a larger portion of the common carotid blood than does the external carotid. In the rabbit, when contrast solutions were injected into the internal carotid artery, blood-brain barrier damage was obtained with contrast solutions only one half as concentrated and of one tenth the volume as those producing damage in the unmodified subject.[23] Heart

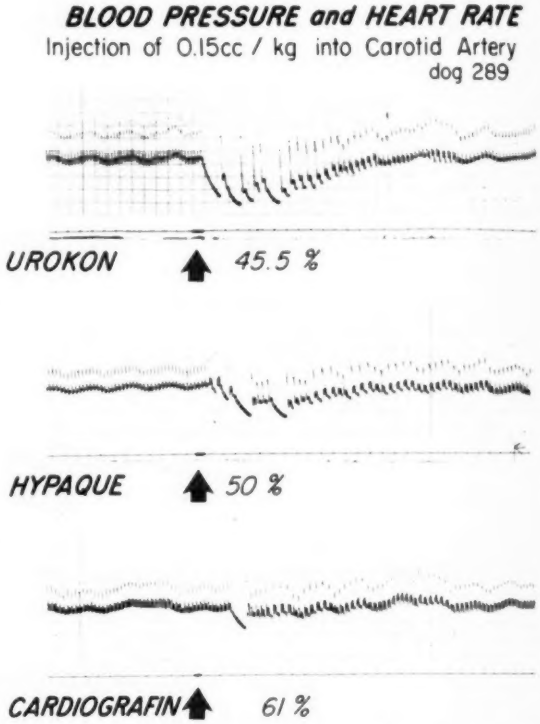


FIG. 8. Comparison of the arterial blood pressure recordings of urokon, hypaque, and cardiografin in dog 289. Equal injection times are indicated by the black bar on the time line.

rate and blood pressure changes of the same nature, as herein reported, have been produced in cats by carotid artery injections of 0.01 to 0.2 cc./kg.[16,17] To sum up, the cardiovascular responses are not only sensitive indications of toxicity, but ones based upon doses comparable to those in clinical practice.

Detailed discussion of the intricacies of the mechanism of the fall in heart rate, the arrhythmia, and the fall in blood pressure is not appropriate for this paper. The mechanism is predominantly a vagal response to brain stimulation, by the chemical nature of the contrast agent. The responses are not due to stimulation of the carotid sinus either chemically or by increase of intraluminal pressure, nor are they due to reflexes initiated by the contrast material in the external carotid system.[7,17] The responses are thought to be not dissimilar to those of vagal nature resulting from transcranial electric stimulation in man and dog.[20]

Sectioning of the vagus prevents the major acute responses. Protection from the circulatory disturbances is also available by proper adequate atropinization. Although the peripheral effects are capable of being so blocked, central stimulation is not affected. The outward early indication of trauma to the central nervous system may be concealed, but the actual irritation or damage of cerebral structures continues.

Cardiografin is the N-methylglucamine salt of 3,5 diacetamido-2,4,6 triiodobenzoic acid. Hypaque is the sodium salt of the same compound. Urokon, sodium acetrizoate, is sodium 3 monacetyl-amino-2,4, 6 triiodobenzoate. Miokon diprotrizoate is sodium 3,5 dipropionyl-amino-2,4,6 triiodobenzoate. These four compounds differ only in the nature of the side chains substituted in the 3,5 positions of the benzene ring. The remainder of the structure, the iodinated benzene ring with the iodine atoms at positions 2,4 and 6 are exactly the same. Diodrast, iodopyracet, is the sodium and diethanolamine salts of 5-diido-4 pyridone-N-acetic acid, a substantially different molecule. The chemical structures are shown in Figure 10.

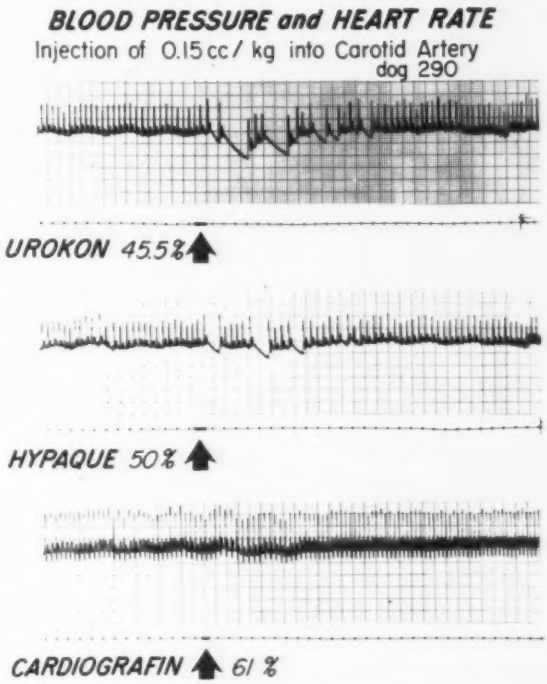
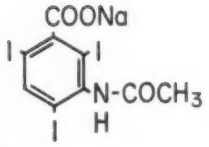


Fig. 9. Comparison similar to that in Figure 8. The disturbances are not as great, but the relative abnormality is the same.

The described disturbances in cardiovascular functions in laboratory animals may be of clinical importance during and following cerebral angiography. It is known they do occur in human cerebral angiography.[10] Unless continuous monitoring were used, they would not likely be detected, since they usually are of brief duration. For several seconds immediately after injection of the contrast material, the heart is slowed or stopped, blood pressure is low, and cardiac output is decreased. A slower passage of the contrast material through the brain could result. The contrast solution would be less subject to rapid dilution by after flowing blood. Lengthening in the time the cerebral vessels are exposed to the contrast material predisposes to other signs of toxicity, such as increase in permeability of the vessels. Lindgren, whose observations were made with a mercury manometer with its characteristic inertia, does not believe the heart and blood pressure changes occur soon enough to prolong the cerebral circulation time. Our studies, made with electronic instruments and very fast paper

UROKON

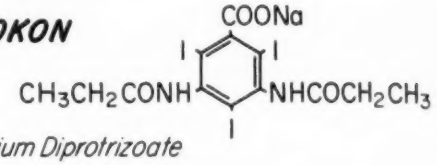


Sodium Acetrizoate

sodium salt of 3-acetylamino-
2,4,6 triiodobenzoic acid

65.8 % Iodine Mol. Wt. 578.9

MIOKON

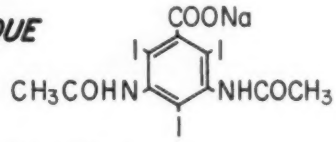


Sodium Diprotrizoate

sodium salt of 3,5 dipropionylamino-
2,4,6 triiodobenzoic acid

57.3 % Iodine Mol. Wt. 663.98

HYPAQUE

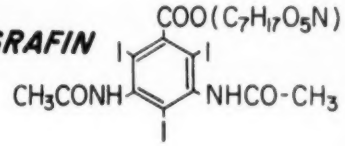


Diatrizoate sodium

sodium 3,5-diacetylamido-
2,4,6 triiodobenzoate

59.87 % Iodine Mol. Wt. 635.8

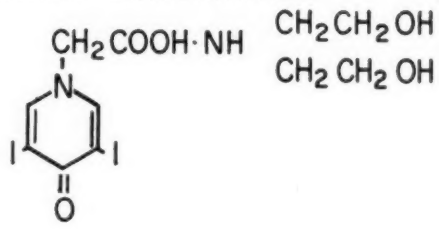
CARDIOGRAFIN



methylglucamine salt of 3,5 diacetylamino-
2,4,6 triiodobenzoic acid

47 % Iodine Mol. Wt. 808.8

DIODRAST



Iodopyracet

3,5 diiodo -4 - pyridone -N- acetic acid diethanolamine

50 % Iodine Mol. Wt. 510

FIG. 10. Chemical structures of the 5 opaque media used in the experiments.

speeds, show the beginning of bradycardia or asystole with accompanying hypotension to occur, at times, before the completion of injection so that they could well have influence on cerebral circulation while the contrast material is on the brain. The elevation of venous pressure would also tend to slow emptying of the cerebral veins and keep contrast material in contact with vascular endothelium for a longer time.

In addition, an effect on the heart itself must be considered. A brief period of hypotension and reduced coronary perfusion may be tolerated by the healthy heart. Morbidity or mortality, however, could result in the already ischemic heart. Although it is realized that most complications of cerebral angiography are neurologic, rarely a patient in the older age group could suffer a coronary occlusion set off by a period of asystole and hypotension.

For adequate testing of contrast materials equal volumes of the same concentration must be injected. In the first experiments, 50 per cent solutions of cardiografin, diodrast, hypaque, miokon and urokon were injected each in single doses of 0.14 to 0.42 cc. per kg. of body weight. In the

later experiments, 50 per cent hypaque was selected as a standard cerebral agent. Hypaque 50 per cent, in wide clinical usage, is considered to have a relatively low incidence of complications. It is one of the newer triiodinated molecules and has good radiopacity. Primarily, radiopacity depends on the iodine content of the molecule. Equating angiographic contrast materials on the basis of their iodine content is most rational, for in this way equal radiopacity can be obtained. The expression of 50 per cent refers to the number of grams of the compound in 100 cc. of liquid, and is not to be confused with the iodine content. Iodine contents of the organic iodide contrast agents vary widely, from 47 per cent for cardiografin to urokon, iodine content 65.8 per cent. The urokon molecule, therefore, has 40 per cent more iodine than cardiografin.

For this reason, cardiografin and urokon were tested in concentrations in which their iodine contents were equated to hypaque 50 per cent (hypaque has an iodine content of 59.9 per cent, miokon 57.3 per cent, diodrast 50 per cent). Sixty-one per cent cardiografin and 45.5 per cent urokon have iodine contents equal to 50 per cent hypaque.

If the objection is made that a lesser concentration than 50 per cent will still afford satisfactory radiopacity clinically, the present studies remain valid. The experiments tested the toxicity of the different molecules on the basis of chemical concentration or on radiopacity. A 50 per cent solution was convenient as a standard because it gave measurable response, and was also in use clinically. Since some radiologists find 30 per cent urokon or 35 per cent diodrast sufficiently opaque, then cardiografin of iodine content equated to these compounds should cause even less disturbance in cardiovascular functions. Forty-two per cent cardiografin equals 30 per cent urokon in iodine content; 37 per cent cardiografin is equivalent to 35 per cent diodrast. Toxicity is no doubt reduced with lower concentrations and smaller volumes of any of the contrast agents, although we did not test solutions in the 30–35 per cent range. The important point remains, for equal radiopacity, cardiografin is the least toxic. Hypaque is intermediate, and urokon the most toxic. For cerebral angiography we advise the least toxic compound at the lowest concentration to give satisfactory opacity to the vessels. We do not believe organic iodides can be used in concentration low enough to avoid all toxic manifestations and yet offer clinically acceptable radiopacity.

Another approach to toxicity of cerebral angiographic contrast materials has been the administration of protective substances prior to injection. Glucose and procaine had been found effective in protecting the dogs' spinal cord from the damaging effects of urokon in experimental aortography.[15] When an attempt was made to use the two substances in clinical carotid arteriography, the prior intracarotid injection of procaine caused seizures in 3 of 8 patients. Administration of glucose prior to angiography did not greatly disturb the patients, but resulted in diminished roentgenographic contrast of the internal carotid system.[24] Until a more safe and reliable means of protecting against the toxic effects of a contrast material can be found, the compound with the fewest and most minimal toxic effects must be sought for and used.

Table 11 lists the previous experiments assessing the toxicity of contrast agents for cerebral angiography. The results closely parallel those of the present work, with few exceptions, despite a variety of methods used, and a wide dose range. Neither the present work nor the previous experiments listed in Table 1 can be taken as unequivocal evidence of the superiority of one contrast material over another for clinical usage. Despite pharmacologic inertness and lack of toxicity in animals, a contrast material may still give rise to complications and side effects in man. It is reasonable to assume, however, a consistent lack or paucity of toxic side effects, as based on several different criteria in animals, is a strong point in favor of a particular contrast material. We are not aware of published data

TABLE II

COMPARISON OF CEREBRAL ANGIOGRAPHIC CONTRAST MEDIA

Laboratory Method of Evaluation	Measurement of Toxicity—Degree of Derangement				
	Least ←	Intermediate			→ Most
Blood-brain-barrier permeability after carotid arteriography (Whiteleather and DeSaussure[27])	Hypaque 50%	Diodrast 35%	Urokon 30%	Urokon 50%	
Functional and anatomic changes after lumbar aortography (Tindall *et al.*[25])	Renografin 70%	Hypaque 70%	Miokon 70%	Diodrast 70%	Urokon 70%
Coronary blood flow, myocardial contractility, electrocardiogram in coronary arteriography (Guzman and West[11])	Diodrast 70%	Renografin 76%	Cardiografin 85%	Hypaque 50%	Urokon 70%
Blood-brain-barrier permeability, neurologic and respiratory changes after carotid arteriography (Winthrop-Stearns, Inc.[28])	Cardiografin 50%	Hypaque 50%	Diodrast 50%	Urokon 50%	
Cardiovascular disturbances in carotid arteriography (Lindgren *et al.*[16–18])	Hypaque 50% Miokon 50%			Triurol 50%	
Blood flow after peripheral arteriography (Lindgren and Törnell[18])	Hypaque 45%			Urokon 25–70%	
Cerebral vascular disturbances after carotid arteriography (Schmidt[22])	Perabrodil 45% Thorotrast Joduron 30% Urografin 40%			Perabrodil M 80% Joduran 70% Urografin 76%	
Blood-brain barrier permeability, histopathologic and electroencephalographic changes and seizures after carotid arteriography (Bloor *et al.*[2])	Diodrast 30–70%			Urokon 30% Neolopax 37.5%	
Blood-brain-barrier permeability after carotid arteriography (Broman and Olsson[4–6])		Triurol 25% Umbradil 35%	Diodrast 35%		
Cerebral blood flow after carotid arteriography (Kågström *et al.*[13,14])	Hypaque 50% Miokon 50%	Umbradil 50%	Triurol 50%		

on extensive use of cardiografin in clinical cerebral angiography. The evidence from animal studies indicates the worthiness of an adequate clinical trial for this agent. Fewer complications and side effects for man are expected.

SUMMARY

Cerebral angiographic contrast media have been compared on the basis of their cardiovascular effects, after intracarotid injection in experimental animals. The abnormalities in heart rhythm, arterial and venous pressure, and cardiac output are considered a measure of the toxicity of a contrast medium. These disturbances may also be of clinical importance. Cardiografin caused the least disturbances, urokon the most, while hypaque was intermediate in effect.

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AN IMPROVED METHOD IN DIAGNOSTIC ROENTGENOGRAPHY OF THE BREAST*

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SOFT tissue roentgenography of the female breast is neither a new nor unproved procedure. The examination, until very recently, has been utilized only by a few men. Leborgne,[13,14] Gros and Sigrist,[10] Gershon-Cohen[3–9] and others[11,12] have done much in developing the technique of breast roentgenography for diagnostic purposes. Egan,[1] in a soon to be published paper, states that he has analyzed 1,000 cases with less than a 1 per cent error. In his series the smallest demonstrable malignant lesion was 2.0 mm. in diameter. The fact that he diagnosed 19 cases of unsuspected or nonpalpable malignancies emphasizes not only the value of this examination in clinical use but suggests its merit as a survey procedure.[6,7,8]

The use of very fine grain industrial x-ray film has made possible roentgenograms of superior quality. Very small lesions are demonstrable and the characteristic calcifications within malignancies are seen in a much greater percentage of cases. The detail of the normal and the abnormal breast structure has been so improved that an accurate diagnosis is possible in most cases.

TECHNIQUE

At the Pennsylvania Hospital we have employed the method advocated by Egan and have attained comparable accuracy in diagnosis. Our cases are unselected and much the same as would be seen in any general hospital. We feel that breast roentgenography can be adeptly performed by any radiologist once he becomes familiar with the various normal and abnormal appearances. The period of self-training is not excessive; however, he should have a good knowledge of the gross anatomy and be able to produce roentgenograms of good quality.

Both breasts are roentgenographed in the superior-inferior and tangential planes. In this manner areas of abnormality are accurately localized. Type M Eastman industrial x-ray film is used in cardboard holders. The physical factors are quite variable, but range from 28 kv. and 400 mas. for the small, atrophic breast to 38 kv. and 700 mas. for very large and secretory or fibrous structures. The primary beam is unfiltered.

All patients are examined by a radiologist who records the physical findings and pertinent history, but the roentgenograms are first reviewed by another radiologist who has not seen the patient. In this manner the provisional interpretation is not influenced by any preconceived opinion. The final report is made when both radiologists, after a discussion of the roentgenographic and physical findings, concur in the diagnosis.

INDICATIONS

Breast roentgenography is not offered as a substitute for breast biopsy. It is of definite value, however, and indicated for: (1) the examination of large, pendulous breasts where palpation is notoriously inaccurate; (2) the detection of nonpalpable masses; (3) the detection of malignant change in breasts containing areas of chronic mastopathia cystica; (4) the disclosure of malignant changes in masses thought clinically to be benign; (5) the detection of the presence of bilateral breast malignancy in cases where only a unilateral mass is palpable; and (6) as a baseline record in cases of known mastopathia cystica. In these

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women periodic breast roentgenography is of extreme value because malignant lesions can long escape detection by palpation.

MATERIAL

At the Pennsylvania Hospital, breast roentgenography has been performed in a total of 206 cases. There have been 34 cases of carcinoma diagnosed by this method of which 32 have been proven correct. In 2 cases breast biopsies were not obtained and the histologic diagnosis is unknown. The first of these women had diffuse metastatic disease and the breast roentgenograms showed a typical carcinoma, but it was deep in an extremely large and fatty structure and not palpable. Because of this the surgeon did not wish to undertake a biopsy procedure. The ovaries, however, were available for histologic examination and were found to be almost completely replaced by tumor compatible in appearance with breast carcinoma.

The second woman had a palpable mass, edema and inflammation of the skin of the breast of two weeks' duration. The breast roentgenograms revealed marked increase in the density of the involved breast, indicative of edema of the breast tissue and skin. No mass was visualized. The proposed differential diagnosis was acute mastitis or inflammatory carcinoma. The mass and edema regressed within one week and only heavy cords of tissue were then palpable. This patient died recently of a cerebrovascular accident, but permission for autopsy or biopsy was not granted. It is felt that this woman had acute mastitis which regressed completely inasmuch as there was no palpable tumor or evidence of tumor metastases at the time of death.

One case of breast carcinoma escaped detection roentgenographically. There was considerable mastopathia cystica and later a carcinoma less than 2.0 mm. in diameter was found in an area most marked by fibrosis. It was detected only after multiple permanent tissue slides were made. A review of the breast roentgenograms failed to show any changes indicative of this malignancy. It was not suspected by the surgeon at the time of the operation.

In another case there was a 1.0 by 1.5 cm. lesion that was considered suspicious, but not diagnostic, of carcinoma. Biopsy revealed this to be fibrous tissue. In view of our present experience we would no longer suspect such a lesion of being malignant.

There have been 4 patients with nonpalpable breast malignancies in whom the

Fig. 1. Case 1. Normal postmenopausal breasts. *Top:* lateral tangential view. *Bottom:* superior-inferior view.

FIG. 2. Case II. Large cystic masses are visualized in both breasts. *Top:* superior-inferior view. *Bottom:* tangential view.

lesions were correctly diagnosed by breast roentgenography.

Of the benign lesions, one woman exhibited bleeding from the nipple but no palpable mass. Roentgenograms showed some minimal increased density in the subareolar tissues, but nothing indicative of malignancy. On the basis of the essentially negative roentgen appearance and the history of bleeding, a diagnosis of intraductal papillomatosis was made, which proved to be correct.

Another woman exhibited a swollen and tender breast. Roentgenograms showed edema of the breast tissues and skin, but no mass. In view of the previous case in which similar findings were present, the correct diagnosis of acute mastitis was made.

In still another woman in whom there had been direct trauma to the breast three years previously, a large cystic mass was visualized. It had only benign features and was thought to be a hematoma. Biopsy was refused.

FIG. 3. Case III. Bilateral fibroadenomata. Smaller tumor on the left was not palpable.

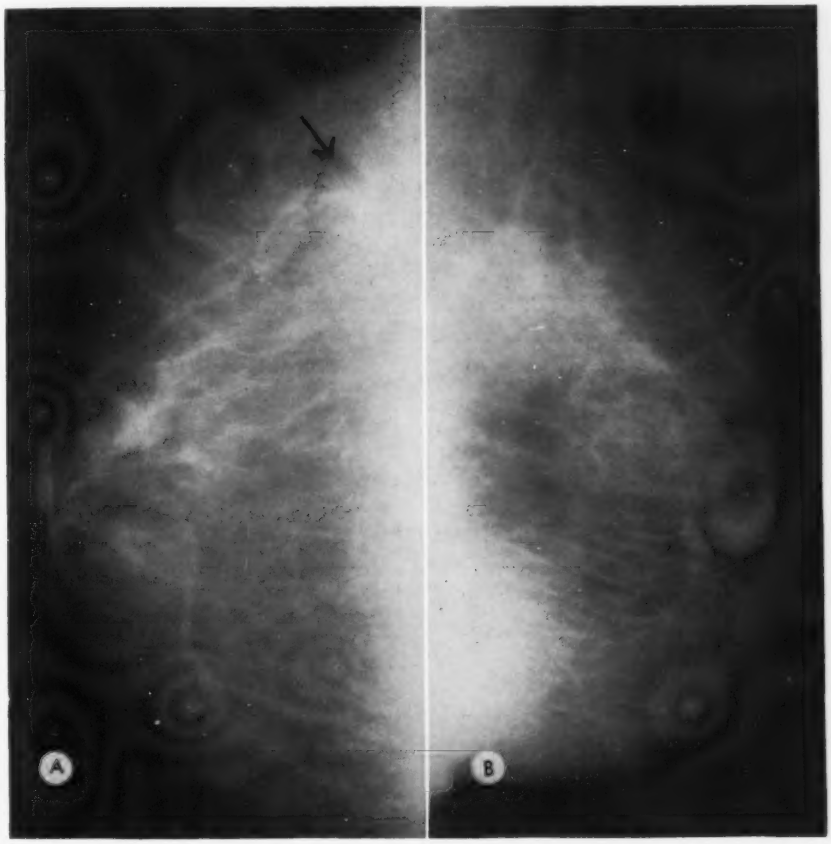

Fig. 4. Case IV. (*A*) Dense, irregular mass (arrow) typical of carcinoma. Superior-inferior view. (*B*) Note the carcinoma with radiating spicules of tumor tissue and increased vascularity. Tangential view.

ERRORS IN DIAGNOSIS

The known errors in the roentgenographic diagnosis of breast lesions in our series include one of commission and one of omission. In a third case we were unable to distinguish between acute mastitis and inflammatory carcinoma.

DIFFICULTIES IN DIAGNOSIS

The roentgenograms of secretory breasts of premenopausal women are quite difficult to interpret.[5] The tissues are very dense and irregular. Sclerosing adenosis with areas of punctate calcifications is impossible to distinguish from carcinoma.[11] Acute mastitis and inflammatory carcinoma have the same roentgen appearance and can be differentiated only when a mass with malignant characteristics is demonstrated.

Specific diagnosis is easier and more accurate in the cases of atrophic or fatty breasts of postmenopausal women. Mastopathia cystica and fibroadenomata should pose no difficulty in diagnosis.[2,3]

All cases in our series have not been proved by histologic examination since both clinically and roentgenographically many were felt to be nonsurgical conditions; however, those cases which were clinically suspected of being malignant have been biopsied and correct diagnoses have been made.

ILLUSTRATIVE CASES

Case I. This forty-four year old woman complained of pain in the left breast. She had an artificially induced menopause several years previously. The roentgenograms of the breasts (Fig. 1) demonstrated normal postmenopausal structures. There was a rather pronounced radiolucency due to fatty tissue. The normal

septa and blood vessels were well visualized and no masses or calcifications were noted.

CASE II. This forty-six year old premenopausal woman had had mastopathia cystica for many years. At the time of roentgen examination she presented bilateral cystic masses which were easily palpable. These were quite painful. Roentgenograms demonstrated severe mastopathia cystica (Fig. 2). Large, dense cysts were present bilaterally as well as clusters of fibrous tissue. The cysts were fluid-filled and their borders were sharp, even though they were lobulated in contour.

CASE III. This thirty-one year old premenopausal woman presented herself because of a mass in the right breast. Roentgenograms (Fig. 3) showed very dense, sharply demarcated masses typical of fibroadenomata.[2] The smaller mass on the left was not recognized by the surgeon at the time of manual examination.

CASE IV. This sixty-six year old woman palpated a hard mass in her breast. The examining physician felt that, clinically, it was malignant. Roentgenograms of the involved breast (Fig. 4, *A* and *B*) showed a very dense, irregular mass in the upper outer quadrant. Spicules of tumor tissue radiated into the surrounding breast substance and there was marked increase in the number and size of the blood vessels in and about the tumor. The appearance was typical of carcinoma.

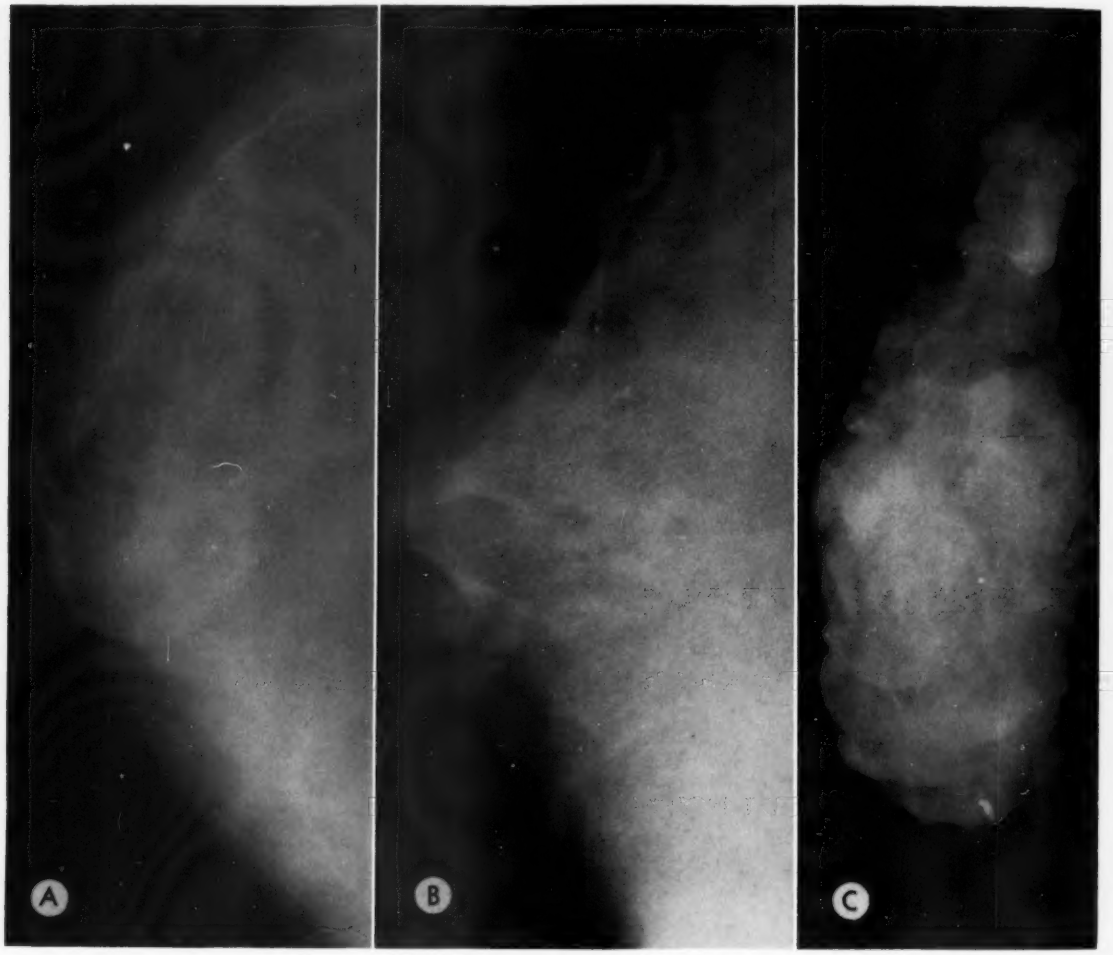

FIG. 5. Case V. (*A*) Centrally located, irregular area of calcification. Superior-inferior view. (*B*) Large calcification and smaller areas of punctate calcifications are demonstrated. The malignant mass lies deep to the calcifications. (*C*) Resected breast mass showing large area of calcification (bottom) and many smaller calcifications.

Case v. This fifty-eight year old postmenopausal woman had an easily palpable 2.0 cm. lymph node in the right axilla. It was smooth, moderately firm and unattached to either the superficial or deep structures. It was neither painful nor tender. Repeated examinations of both breasts over a period of two months failed to reveal any palpable tumor. Roentgenograms (Fig. 5, *A* and *B*) demonstrated a large area of irregular calcification and several small punctate calcifications.[4] An ill-defined, extremely irregular mass was visualized deep to these calcifications. This type of calcification is indicative of malignancy.[4,13] Roentgenograms of the axillary lymph node showed it to be very dense and smooth. This, too, indicated malignant tissue content. The resected tumor mass (Fig. 5*C*) revealed the large area of calcification as well as many smaller calcifications. Subsequent histologic examination proved the calcifications to be areas of necrotic tumor. The roentgenographically dense axillary lymph node was the only one containing metastatic tumor.

SUMMARY

Diagnostic breast roentgenography is a valuable and accurate procedure which can be performed by utilizing roentgenographic equipment available in most radiologic departments or offices. The use of very fine grain industrial x-ray film ensures roentgenograms of superior diagnostic quality. The accuracy of this method is well documented in both the series of Egan[1] and that of the authors. Out of 206 cases examined at the Pennsylvania Hospital, 34 were diagnosed as carcinoma; of these 32 have been proven to be correct. The roentgenograms of 5 patients are presented.

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A DEVICE TO FACILITATE THE INTRODUCTION OF A CATHETER INTO THE TRACHEA FOR PASSIVE BRONCHOGRAPHY*

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ONE of the chief handicaps in performing passive bronchography is difficulty in passing the catheter into the trachea. Usually this is accomplished by inserting the catheter through the nose after the hypopharynx has been anesthetized by topical anesthesia. All too often the catheter is expelled from the trachea because of coughing, and the tip is deflected posteriorly into the esophagus. It appeared desirable, therefore, to devise an instrument through which passage of the catheter into the trachea could be performed easily under fluoroscopic control, and which

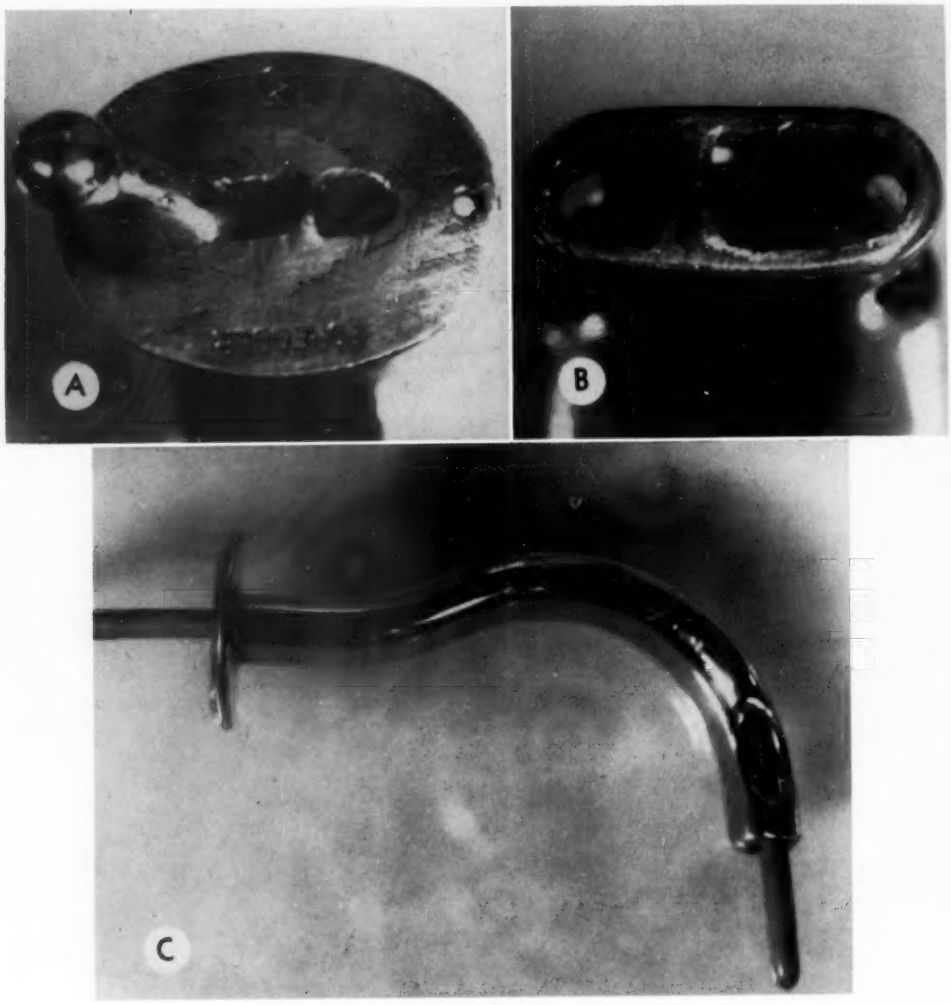

FIG. 1. (*A*) Proximal end of adapted airway. (*B*) Distal end of adapted airway. (*C*) Catheter threaded through airway.

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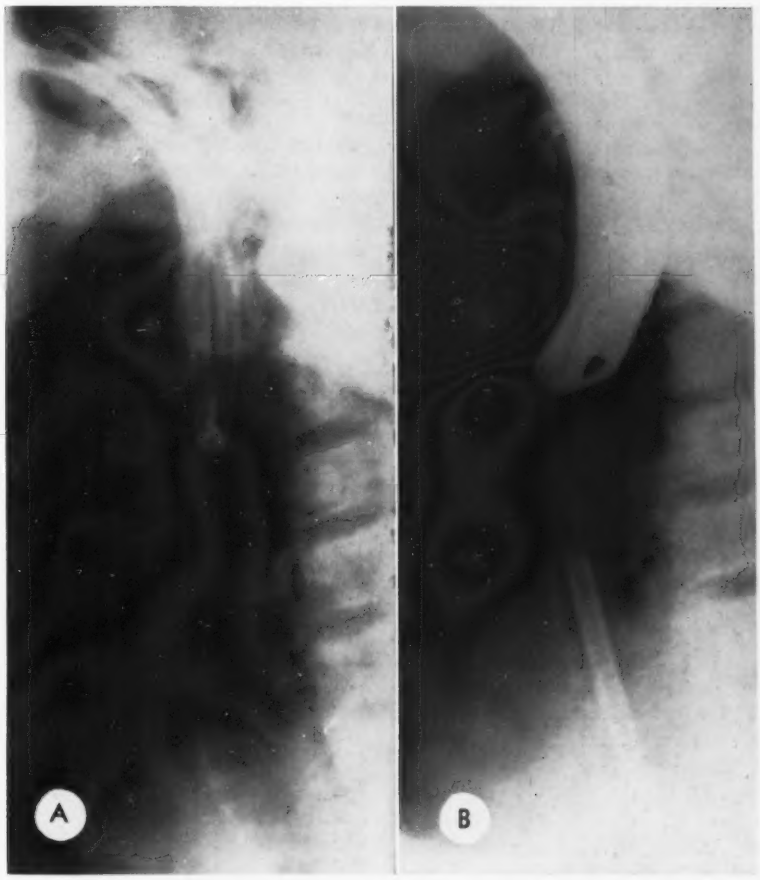

Fig. 2. (*A*) Spot roentgenogram showing catheter introduced through the nose. The tip of the catheter has turned back upon itself and is in the patient's mouth. (*B*) Catheter introduced through adaptor airway. The entrance into the trachea was rapid, and even though the patient coughed violently, the catheter was not displaced from the trachea.

would help keep it there as long as indicated.

To accomplish this, a Foregger No. 5 airway, in which a suction tube is incorporated, was adapted by welding two small bars at its proximal and distal ends, thereby establishing a guiding passageway through which a No. 14 French urethral catheter could be passed. The device is placed in the patient's mouth after local anesthesia has been applied, and under fluoroscopic control, its tip is directed towards the pharyngotracheal junction. The tip of the catheter is placed directly over the larynx, the patient is asked to breathe deeply, and the catheter is then introduced into the trachea. When this has been accomplished, a small quantity of pontocaine or cocaine is introduced into the catheter to anesthetize the carina. The study can then proceed without fear of the patient coughing back the catheter, with the confidence of having an adequate airway in place through which the catheter can be positioned as desired, and to which suction can be applied if necessary.

Long Island Jewish Hospital
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THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE

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Editor: Lawrence Reynolds, 110 Professional Building, Detroit 1, Mich.

Sixty-second Annual Meeting: Hotel Deauville, Miami Beach, Florida, September 26–29, 1961.

* Deceased.

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Forty-fourth Annual Meeting: Waldorf-Astoria Hotel, New York, N. Y., April 1–4, 1962.

* *Ex officio.*

EDITORIALS

THE SIXTY-SECOND ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

"WHAT a beautiful view! . . ." exclaimed awed Astronaut Alan B. Shepard, Jr., in his burst of enthusiasm upon taking his first periscopic glimpse of the land underneath the rising, arcing space ship "Freedom 7." Later, he said: "I was able to see very clearly, of course, The Florida Peninsula, Lake Okeechobee and identify Andros Island (one of the Bahamas) . . . ."

It is a fortunate coincidence that radiologists, who always have been in the vanguard of pioneering and unceasingly fostering protection against the hazards of man-made and space-made radiations, should meet on this "beautiful" peninsula, in the year of such an epoch-making event. The Sixty-second Annual Meeting of the American Roentgen Ray Society will be held, September 26-September 29, 1961, at the Deauville Hotel, in Miami Beach, the pearl of Florida.

In 1895, Röntgen made his famous discovery of the x-ray, inaugurating the era of man-made radiations. In 1896, Bequerel presented to the Paris Academy of Sciences his observations on the radioactivity of uranium compounds, initiating the era of study of the earthborn radiations, which now is rapidly expanding to the spaceborn radiations. In the spring of 1896, Henry M. Flagler, the entrepreneur, drove his puffing train through the subtropical wilderness into a tiny fishing village, laying the foundation of Miami. During the intervening six and one-half decades progress has been swift, tremendous, fabulous: swift in the application of the man-made radiations; tremendous in the understanding of earthborn and spaceborn radiations; fabulous in the full exploitation of human free enterprising ingenuity.

The isolated Miami of 1896 has literally gone "from rags to riches" by 1961, and Miami Beach has become the greatest resort city of the nation with a fantastic skyline of hotels, resplendent in ocean beaches, sands, swimming pools, magnificent gardens, and innumerable tourist attractions of the most fascinating sort. The Deauville Hotel—on the Ocean at 67th Street—in the Center of this skyline, is unique in beauty of lay-out, architecture and decor; in its modern facilities for efficiently running the Annual Meeting; and in its irresistible inducements to resort-type relaxation, pleasure and entertainment.

The forthcoming meeting will be the first since the Society was founded in 1900 to be held in Florida and Miami Beach beckons as an eminently suitable locale. The Committee on Local Arrangements under the able Chairmanship of Dr. Gerard Raap spared no time or effort in planning and organizing the many local details in the truest spirit of southern hospitality.

A preliminary program of the meeting appears elsewhere in this issue of the JOURNAL. The registration desk will be open in the Upper Convention Lobby from 2:00 P.M. to 5:00 P.M. Sunday, September 24 and thereafter daily from 8:00 A.M. to 5:00 P.M. It will close at 1:30 P.M. on Friday, September 29.

The Annual Golf Tournament for members and guests will be held at the La Gorce Country Club, Monday, September 25. The Willis F. Manges Trophy, for Eighteen Hole Medal Handicap, and the Exhibitors' Trophy, for Eighteen Hole Gross Score, and

other prizes will be awarded. The Annual Golf Dinner will follow at the La Gorce Country Club at 7:30 P.M. Arrangements have been made by the Local Golf Committee, under the Chairmanship of Dr. Virgil H. Pieck, for bus transportation to and from the Country Club, and for luncheon, golf and dinner.

The Annual Meeting of the Society will be called to order by President Harold G. Reineke at 8:30 A.M. Tuesday, September 26, in Napoleon Room No. 3. The Address of Welcome will be given by Dr. W. L. Fitzgerald, President of the Dade County Medical Association and Clinical Professor of Urology of the University of Miami, Florida. President Reineke then will install, with the assistance of Dr. Ralph M. Caulk, Chairman of the Executive Council, President-Elect Traian Leucutia as the new President of the Society. Following the Inaugural Address of President Leucutia, the Scientific Sessions will begin.

As in former years, these sessions will be held in the morning so as to afford ample time for attendance of the Instruction Courses in the mid-afternoon and for visits to the Technical Exhibits until 5:00 P.M. and the Scientific Exhibits until later in the evening.

The Program Committee has selected 48 papers to be presented at the Scientific Sessions. An impressive number of these papers encompasses topics dealing with various special procedures, bringing into sharp focus the modern trend in diagnostic roentgenology. As an appropriate expression of this trend, Dr. Harold O. Peterson, who on Tuesday evening at 8:15 will deliver in the Casanova Room this year's Caldwell Lecture, a notable event at all Annual Meetings, has chosen as his subject "The Radiologist and the Special Procedures." Dr. Peterson who is well known to members of the Society as the untiring Director of the Instruction Courses is Chairman of the Department of Radiology of the University Hospitals and Professor of Radiology of the University of Minnesota, Minneapolis, Minnesota. No one speaks with more authority on the chosen subject than does Dr. Peterson and his Lecture undoubtedly will create the greatest interest. Dr. Peterson will be introduced by Dr. J. Paul Medelman, Director of the Department of Radiology, St. Joseph's Hospital, St. Paul, Minnesota, and Clinical Associate Professor of Radiology of the University of Minnesota, a distinguished member of the Society of many attainments. The Caldwell Medal will be presented to Dr. Peterson at the conclusion of his Lecture.

Emphasizing the trend toward special procedures, valuable papers will also be presented illustrating the influence of some isotope techniques on diagnostic roentgenology.

The Friday morning Scientific Session will be devoted entirely to therapeutic problems, reflecting the more recent achievements in physics and biologic and clinical progress as well as calling attention to the hazards which may be encountered.

The Director of Instruction Courses, Dr. Harold O. Peterson, assisted by Dr. Donn G. Mosser, Associate Director, has, as usual, assembled a very representative faculty to give these courses, several of which will deal with special procedures. The detailed curriculum of the courses and a list of the members of the Faculty will be published in the August issue of the JOURNAL.

The Committee on Scientific Exhibits, of which Dr. Charles O. Heilman is the Chairman, reports that the increasing interest of the past few years continues and that the Scientific Exhibits are expected to be most instructive and of the highest quality. Several of them accompany or supplement the essays on special procedures.

The number of Technical Exhibits promises to be the largest in the Society's history, fully utilizing the available facilities, despite the fact that Dr. James C. Cook, Manager of the Annual Meeting and Mr. Clifford L. Sherrat, Honorary Member, provided additional space over that of last year's record breaking meeting in Atlantic City. In an era of great technologic prog-

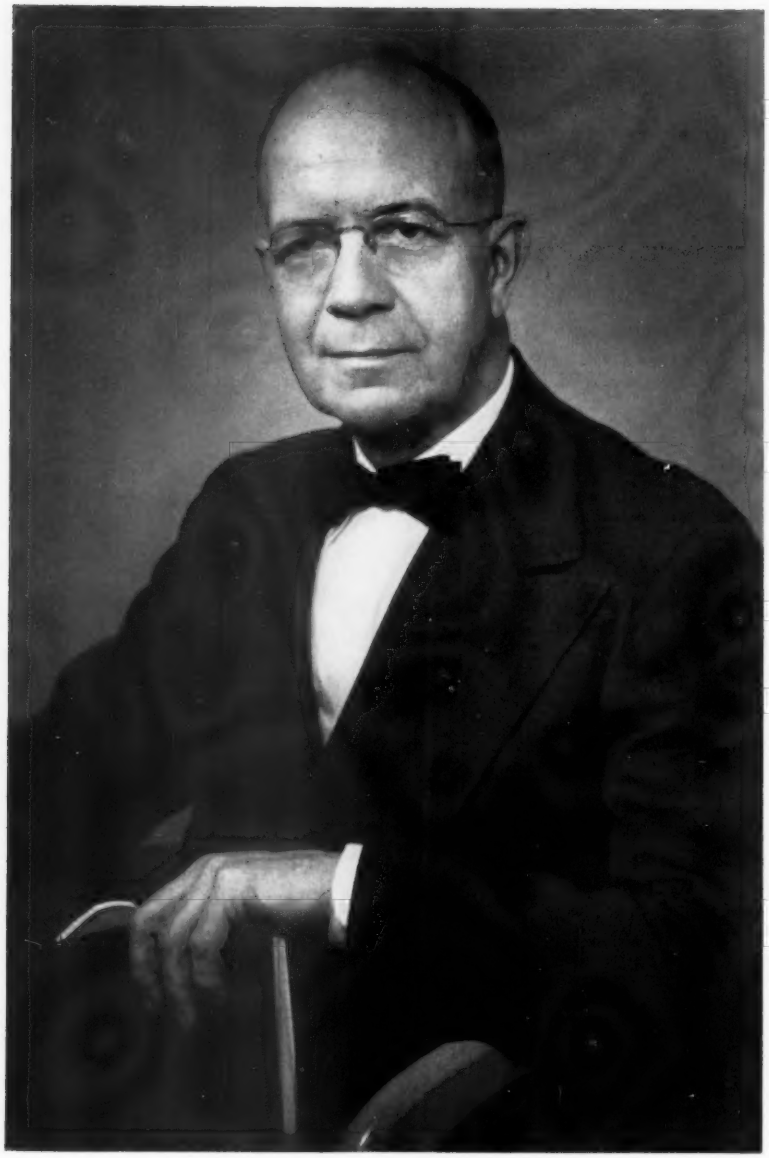
ress, the Technical Exhibits represent an indispensable contribution to the success of the Annual Meeting and to enable visual documentation of the latest accomplishments, they are placed in the immediate vicinity of the Scientific Sessions.

A famous resort city, such as Miami Beach, affords unusual opportunities for social activities of the most delightful variety. This is particularly evident at this year's meeting of the American Roentgen Ray Society. The Annual Banquet which will be held Thursday evening will feature a "Nite in Caribbean." At 6:30 P.M. there will be a Cocktail Hour in the Pool Area with Music by Count Ivan and his Calypsonians. At 7:30 P.M. the banquet will start in the Casanova Room. There will be dinner dancing to the tunes of the Murray Carmen Orchestra, followed by Presentation of the 1961 Scientific Awards and an exclusive "Islander" Floor Show.

The Ladies' Committee on Local Arrangements under the able and enthusiastic Chairmanship of Mrs. Gerard Raap and Mrs. J. S. Stewart has arranged a splendid program for the ladies. On Monday, leaving the hotel at 3:30 P.M., there will be a Golf Widows' Boat Cruise to Fort Lauderdale, with Dinner at Pier 66 at 6:00 P.M., and returning by air-conditioned buses to the hotel at about 10:00 P.M. On Tuesday, starting at 10:00 A.M., there will be a Bus Tour to the Parrot Jungle, lunch at Jamaica Inn and a visit to the Seaquarium, with return to the hotel at 4:00 P.M. Wednesday will be occupied by a boat trip to Viscaya, with lunch en route, and a visit to the historic Villa Viscaya, renowned for its rare art treasures, extensive formal gardens and the many pools and fountains adorned by classic sculptures. A visit to this amazing place alone is worth a trip to the Annual Meeting. The daytime of Thursday was left open for extraprogrammatic activities and the evening was reserved for a "Nite in Caribbean."

The Officers of the American Roentgen Ray Society extend a cordial invitation to its members, all radiologists and their guests, along with their wives and families, to attend this meeting in the fabulous city of Miami Beach on the "beautiful" Florida Peninsula.

Traian Leucutia, M.D.
President-Elect
Harper Hospital
Detroit, Michigan



FRANKLIN B. BOGART
1894–1961

ON March 11, 1961, Dr. Franklin B. Bogart of Chattanooga, Tennessee, died at the age of sixty-six of a chronic valvular heart disease. Although he had limited his activities, he continued daily visits to his office until a few days before his death.

Franklin Bogart was born in Chattanooga, Tennessee, the son of Dr. William Moore Bogart and Keturah Thompson Bogart. He was named for his grandfather, Dr. Franklin Bogart, who practiced medicine in upper middle Tennessee. In 1916 he was graduated from the University of Wisconsin with a B.S. degree and received

his M.S. degree from the University of Wisconsin in 1917. He was graduated from Johns Hopkins University with an M.D. degree in 1919, and returned to Chattanooga in that year to establish his practice. Dr. Bogart was first associated with Dr. W. H. Cheney, who was a pioneer radiologist in this area. In 1920, Franklin Bogart married Rose Alice Harloff. He continued in the private practice of Radiology in Chattanooga until 1942, at which time he entered military service, serving as Major and later as Lieutenant Colonel in the Medical Corp AUS, as Chief of the Radiological Branch of the Lettermann General Hospital in San Francisco, Clifornia.

An ardent student of radiology and a loyal worker for organized medicine, he was a Fellow of the American College of Radiology and a Fellow of the American College of Medicine, a Diplomate of the American Board of Radiology, a member of the American Radium Society and the Radiological Society of North America, a member of the American Roentgen Ray Society and Vice-President of that organization in 1959, President of the Chattanooga and Hamilton County Medical Society and President of the Tennessee State Medical Association. He was particularly active in affairs of the Cancer Society in the State and was Co-Director of the Chattanooga Tumor Clinic, Inc., Vice-President of the Hamilton County Unit of the Tennessee Division of the American Cancer Society and served on the Board of Directors of both the local unit and the Tennessee Division for many years.

Dr. Bogart was a member of the Lookout Mountain Presbyterian Church and a member of the Fairyland Club of Lookout Mountain. He was a Rotarian and exemplified the motto, "Service above Self," in his daily practice.

Dr. Bogart was an enthusiastic gardener and in the spring the azaleas and dogwoods that thrived in his extensive gardens were a thrilling site to visitors and to passersby. In recent years, he had become interested in orchid culture and his hothouse yielded many beautiful specimens.

He is survived by his wife, Rose, and by his daughters, Alice Bogart and Mrs. David S. Smith of Westport, Connecticut, and two granddaughters.

Dr. Franklin Bogart will long be remembered as an outstanding radiologist and a loyal and honorable physician, highly respected by all of his colleagues, and will be greatly missed by them, his patients, his family and his many friends.

George K. Henshall, M.D.

DAVID M. GOULD
1914–1961

ON April 2, 1961, with the sudden death from myocardial infarction of Dr. David M. Gould, many of us lost a good friend and colleague.

Dr. Gould was born in Worcester, Massachusetts, on January 29, 1914. He attended Classical High School in Worcester and received his A.B. degree from Clark University in 1935. Upon graduation from college, he entered the Harvard Medical School where he was awarded his M.D. degree in 1939. After serving as an intern at the Boston City Hospital from 1939 to 1941, he was commissioned a Lieutenant in

the United States Public Health Service.

During the war years Dr. Gould saw service in Europe, attaining the rank of Surgeon (Major). He was assigned to duty with the United Nations Relief and Rehabilitation Administration in 1945, and in 1947 was sent to The Johns Hopkins Hospital to pursue further training in Radiology. He was appointed to the faculty of The Johns Hopkins University as an Assistant Professor in 1949, and was promoted to Associate Professor in 1950. During his years at Johns Hopkins he won the universal respect and regard of his colleagues for his keen clinical judgment and for his fine personal qualities.

In 1955 he was an exchange Professor at Guy's Hospital Medical School in London, and he lectured at Edinburgh Medical School in Scotland and at Lund University Medical School in Sweden.

In 1956, Dr. Gould was appointed Professor and Head of the Department of Radiology at the New University of Arkansas Medical Center in Little Rock. He remained there until September, 1959, at which time he came to the University of Colorado as Professor and Head of the Department of Radiology. In his three years at the University of Arkansas he developed an outstanding department which was widely recognized for its teaching, service, and research programs. At the University of Colorado, Dr. Gould again exhibited his remarkable leadership. He was particularly effective in attracting able young men to the field of Radiology, and the loyalty of all his staff, both professional and nonprofessional, testified to his leadership.

As a teacher, Dr. Gould inspired a host of students and residents by his keen judgment and modest personality. He was a Diplomate of the American Board of Radiology and a Fellow of the American College of Radiology (Alternate Counselor to the College from Colorado, 1960) and of the American College of Chest Physicians. He was a member of the Radiological Society of North America; the American Roentgen Ray Society; the Colorado State Medical Society; the American Medical Association; the American Association of University Professors; the American Association of University Radiologists (Past-President); the American Association for the Advancement of Science; the American Society of Nuclear Medicine; the Colorado Radiological Society; and the Rocky Mountain Radiological Society.

His many friends throughout the country join with his family and his Colorado colleagues in mourning the passing of a true gentleman and scholar.

John S. Bouslog, M.D.

PRELIMINARY PROGRAM

SIXTY-SECOND ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

The Sixty-second Annual Meeting of the American Roentgen Ray Society will be held at the Deauville Hotel, Miami Beach, Florida, Tuesday through Friday, September 26 to 29, 1961.

The Executive Council will meet Sunday, September 24, 9:00 A.M. in the Normandy Room.

On Monday, September 25, the Annual Golf Tournament for Members and Guests —for the Willis F. Manges Trophy and the Exhibitors' Trophy—will be held at the La Gorce Country Club, followed by the Annual Golf Dinner at 7:30 P.M. at the same place.

The Scientific Sessions will be held in the morning from 8:30 A.M. to 12:30 P.M., Tuesday through Friday in Napoleon Room No. 3. The Caldwell Lecture will be delivered by Dr. Harold O. Peterson on Tuesday evening at 8:15 P.M. in the Casanova Room. Dr. Peterson will be introduced by Dr. J. Paul Medelman.

The Instruction Courses will be held in the afternoon from 3:00 P.M. to 4:30 P.M., Tuesday, Wednesday and Thursday and from 1:30 P.M. to 3:00 P.M., Friday. The detailed plan of the Instruction Courses will be published in the August issue of the JOURNAL.

The Scientific Exhibits will be set up in the Peacock Alley and Upper Convention Lobby. They will open daily at 8:00 A.M. and will close evenings at 8:00 P.M. Tuesday, 9:00 P.M. Wednesday, and 7:30 P.M. Thursday.

The Technical Exhibits will be located in Napoleon Rooms No. 1 and 2, immediately adjacent to the meeting room of the Scientific Sessions. They will open at 8:00 A.M. and will close at 5:00 P.M., Tuesday through Thursday.

Both the Scientific Exhibits and Technical Exhibits will remain open Friday from 8:00 A.M. until 1:00 P.M.

The Annual Banquet will be held Thursday evening at 7:30 P.M. in the Casanova Room, preceded by a Cocktail Hour beginning at 6:30 P.M. in the Pool area.

The Program Committee under the Chairmanship of President-Elect Traian Leucutia has arranged the following program for the Scientific Sessions.

Tuesday, September 26, 1961
Napoleon Room No. 3

8:30 A.M. Call to Order, Sixty-second Annual Meeting: Harold G. Reineke, M.D., Cincinnati, Ohio, President. Address of Welcome: Dr. W. L. Fitzgerald, President of the Dade County Medical Association; Clinical Professor of Urology of the University of Miami, Miami, Florida.

Installation of President-Elect, Traian Leucutia, M.D., Detroit, Michigan, by Harold G. Reineke, M.D., President, Cincinnati, Ohio, and Ralph M. Caulk, M.D., Chairman of the Executive Council, Washington, D. C.

Inaugural Address: President Traian Leucutia, M.D.

9:10 A.M.

Presiding: Traian Leucutia, M.D.
Detroit, Michigan

1. The Significance of Protein Binding of Contrast Media in Roentgen Diagnosis. Elliott C. Lasser, M.D. (by invitation), Pittsburgh, Pa., Richard S. Farr, M.D. (by invitation), Pittsburgh, Pa., Gerald P. Rodnan, M.D. (by invitation), Pittsburgh, Pa., Tak Fujimagari, M.D. (by invitation), Pittsburgh, Pa., and Walter R. Tripp, M.D. (by invitation), Pittsburgh, Pa.
2. Interesting Aspects of Cerebral Angiog-

graphy with Emphasis on Some Unusual Congenital Variations. C. Richard Perryman, M.D., D.Sc. (Med.), Pittsburgh, Pa., C. H. Gray, M.D. (by invitation), Pittsburgh, Pa., and Raymond W. Brust (by invitation), Pittsburgh, Pa.

3. Angiography As an Aid in Determining Resectability of Lung Cancer. D. E. Sanders, M.D. (by invitation), Toronto, Ont., Can., N. C. Delarue, M.D. (by invitation), Toronto, Ont., Can., and George Lau, M.D. (by invitation), Toronto, Ont., Can.
4. Radiographic Aspects of Anomalous Pulmonary Venous Drainage. Melvin M. Figley, M.D., Seattle, Wash., Leon A. Phillips, M.D. (by invitation), Seattle, Wash., Robert A. Bruce, M.D. (by invitation), Seattle, Wash., Warren G. Guntheroth, M.D. (by invitation), Seattle, Wash., and K. Alvin Merendino, M.D. (by invitation), Seattle, Wash.
5. The Cinefluorographic Approach to the Diagnosis of Pericardial Effusion. Joseph Jorgens, M.D., Ph.D. (by invitation), Minneapolis, Minn., and Robert R. Kundel, M.D. (by invitation), Minneapolis, Minn.
6. The Radiographic Manifestations of Aortic Valvular Disease. E. C. Klatte, M.D., (by invitation), Indianapolis, Ind., J. A. Campbell, M.D., Indianapolis, Ind., W. E. Judson, M.D. (by invitation), Indianapolis, Ind., and P. R. Lurie, M.D. (by invitation), Indianapolis, Ind.
7. Cardiac Ventriculography (Transaortic Catheter Opacification of the Left Ventricle). J. Stauffer Lehman, M.D., Philadelphia, Pa., Joseph N. Debbas, M.D. (by invitation), Philadelphia, Pa., and James J. Boyle, Jr., M.D. (by invitation), Philadelphia, Pa.
8. Flexible Tubing for Flow-Guided Cardiovascular Catheterization and Visualization. Charles T. Dotter, M.D., Portland, Ore., and Kurt R. Straubbe, M.D. (by invitation), Portland, Ore.
9. Extra Large Field Angiography Technique and Results. Albert Jutras, M.D. (by invitation), Montreal, Que., Can., Marcel Longtin, M.D. (by invitation), Montreal, Que., Can., and Paul Roy, M.D. (by invitation), Montreal, Que., Can.
10. Physical Aspects of High Pressure Angiography. Fred C. Shipps, M.D., Portland, Ore.

Tuesday Evening, September 26, 1961
Eight-fifteen o'clock
Casanova Room

THE CALDWELL LECTURE

The Radiologist and the Special Procedures

by

Harold O. Peterson, M.D.

Chairman of Department of Radiology of the University Hospitals, and Professor of Radiology, University of Minnesota, Minneapolis, Minn.

Introduction:

J. Paul Medelman, M.D., Director of Department of Radiology, St. Joseph's Hospital, St. Paul, Minnesota, and Clinical Associate Professor of Radiology, University of Minnesota, Minneapolis, Minnesota.

Wednesday, September 27, 1961
Napoleon Room No. 3

8:30 A.M.

Presiding: Clyde A. Stevenson, M.D.
Spokane, Washington

11. Hemoglobin S-C Disease. Joshua A. Becker, M.D. (by invitation), Philadelphia, Pa.
12. Radiographic and Clinical Appraisal of Sickle Cell-Hemoglobin C Disease. Jack Reynolds, M.D. (by invitation), Dallas, Tex.
13. Radiologic Manifestations of Juvenile Rheumatoid Arthritis. William Martel, M.D. (by invitation), Ann Arbor, Mich., John F. Holt, M.D., Ann Arbor, Mich., and James T. Cassidy, M.D. (by invitation), Ann Arbor, Mich.
14. Cineradiographic Studies of Patients with Cervical Spine Fusion. Malcolm D. Jones, M.D. (by invitation), San Francisco, Calif.
15. Anesthesia for Bronchography Utilizing Aerosol-Nebulizer. William Molnar, M.D. (by invitation), Columbus, Ohio, and John Prior, M.D. (by invitation), Columbus, Ohio.
16. Postoperative Pulmonary Atelctasis. J. Pollard, M.D. (by invitation), Boston, Mass., and H. Bendixen, M.D. (by invitation), Boston, Mass.

17. Breast Cancer Detection by Periodic X-Ray Examination—Five Year Survey. J. Gershon-Cohen, M.D., D.Sc. (Med.), Philadelphia, Pa., M. B. Hermel, M.D., Philadelphia, Pa., and Simon M. Berger, M.D. (by invitation), Philadelphia, Pa.
18. Lymphangiography; Its Role in Diagnosis and Therapy. Sidney Wallace, M.D. (by invitation), Philadelphia, Pa., Laird Jackson, M.D. (by invitation), Philadelphia, Pa., and Roy Greening, M.D., Philadelphia, Pa.
19. Renal Involvement in Lymphomas and Leukemias in Adults. Jerome H. Shapiro, M.D. (by invitation), New York, N. Y., G. Craig Ramsay, M.D. (by invitation), New York, N. Y., Harold G. Jacobson, M.D., New York, N. Y., and Charles Botstein, M.D. (by invitation), New York, N. Y.
20. Curable Arterial Hypertension Due to Renal Artery Lesions, Including Fibromuscular Hyperplasia. A. J. Palubinskas, M.D. (by invitation), San Francisco, Calif.
21. The Radioactive Kidney Test As a Screening Procedure in Hypertension. A. Weber, M.D. (by invitation), Boston, Mass., and T. Agard, M.S. (by invitation), Boston, Mass.
22. Coarctation of the Renal Artery with "Notching" of the Ureter: A Roentgenologic Sign of Unilateral Renal Disease As a Cause of Hypertension. Mordecai Halpern, M.D. (by invitation), New York, N. Y., and John A. Evans, M.D., New York, N. Y.
23. The Effect of (Voluntary) Bladder Distention on the Intravenous Pyelogram: An Experimental Study. J. S. Dunbar, M.D., Montreal, Que., Can., D. W. MacEwan, M.D. (by invitation), Montreal, Que., Can., and Bernedette Nogrady, M.D. (by invitation), Montreal, Que., Can.
24. The Radiation Hazard. Gerald M. McDonnel, M.D. (by invitation), Los Angeles, Calif.
25. Opacification Studies of Normal and Abnormal Paranasal Sinuses. Lewis E. Etter, M.D., Pittsburgh, Pa.
26. Roentgen Demonstration of Cranial Nerve Metastases in Tumors of the Head and Neck. Gerald D. Dodd, M.D. (by invitation), Houston, Tex.
27. Intra-aural Radiography of the Fenestra Ovale. O. Arthur Stiennon, M.D. (by invitation), Madison, Wis., and James F. Land, M.D. (by invitation), Madison, Wis.
28. Skeletal Abnormalites Associated with Gonadal Dysgenesis. Nathaniel Finby, M.D., New York, N. Y., and Reginald M. Archibald, M.D. (by invitation), New York, N. Y.
29. Chondromyxoid Fibroma of Bone. Bernard Turcotte, M.D. (by invitation), Rochester, Minn., David G. Pugh, M.D., Rochester, Minn., and David C. Dahlin, M.D. (by invitation), Rochester, Minn.
30. What Is Multiple Myeloma? Robert Phelps Barden, M.D., Philadelphia, Pa.
31. Roentgen Findings in Diseases with Abnormal Calcium Deposition. Theodore F. Hilbish, M.D., Bethesda, Md., and Frederic C. Bartter, M.D. (by invitation), Bethesda, Md.
32. Radiologic Findings in the Small Bowel in Scleroderma. James Reinhardt, M.D. (by invitation), Durham, N. C., and William F. Barry, Jr., M.D., Durham, N. C.
33. Duodenal Roentgenographic Manifestations of Non-Tropical Sprue. John E. Shick, M.D. (by invitation), Detroit, Mich.
34. Fluid-Filled Loops in Intestinal Obstruction. J. L. Williams, M.D. (by invitation), Danville, Pa.
35. The Use of Silicone Foam As an Enema in Examining the Human Sigmoid Colon. Galen B. Cook, M.D. (by invitation), St. Louis, Mo., and Alexander R. Margulis, M.D., St. Louis, Mo.
36. Scintillation Scanning of the Liver. Frederick J. Bonte, M.D., Dallas, Tex., J. S. Krohmer, M.A., Dallas, Tex., Ernest A. Elmendorf, M.D. (by invitation), Dallas, Tex., Norman L. Presley, M.D. (by invitation), Dallas, Tex., and Giles J. Andrews, M.D. (by invitation), Dallas, Tex.

Thursday, September 28, 1961
Napoleon Room No. 3

8:30 A.M.

Presiding: Ralph M. Caulk, M.D.
Washington, D. C.

Friday, September 29, 1961
Napoleon Room No. 3

8:30 A.M.

Presiding: Robert C. Pendergrass, M.D.
Americus, Georgia

37. Analysis of the Results of X-Ray Therapy by Region in Malignant Lymphoma. Lillian M. Fuller, M.D. (by invitation), Houston, Tex.
38. Radiation Therapy of Chronic Lymphatic Leukemia—a Statistical Analysis. James C. Cook, M.D., Detroit, Mich., and Walter Romano, M.D. (by invitation), Detroit, Mich.
39. Results in Squamous Cell Carcinoma of the Cervix: A Comparison of Orthovoltage and Cobalt 60 Radiation. George Cooper, Jr., M.D., Charlottesville, Va., and David H. Williams, M.D. (by invitation), Charlottesville, Va.
40. Evaluation of Complications Encountered in the Treatment of Malignancy with the Telecobalt Beam, with Recommendations for Measures to Avoid Them. Damon D. Blake, M.D. (by invitation), Winston-Salem, N. C., I. Meschan, M.D., Winston-Salem, N. C., and W. A. Covell, M.D. (by invitation), Winston-Salem, N. C.
41. Osteoradionecrosis of the Mandible. Bernard Roswit, M.D. (by invitation), Bronx, N. Y.
42. Dose-Time Relationships in Clinical Radiotherapy; Clinical Observations and Rabiobiological Correlation. J. Robert Andrews, M.D., Bethesda, Md., Kirkland C. Brace, M.D. (by invitation), Bethesda, Md., and Roger J. Berry, M.D. (by invitation), Bethesda, Md.
43. The Second Half-Value Layer and the Homogeneity Factor. E. Dale Trout, B.S., D.Sc., Milwaukee, Wis., John P. Kelly, B.S. (by invitation), Milwaukee, Wis., and Arthur C. Lucas, B.S. (by invitation), Milwaukee, Wis.
44. The Effect of Lowering Temperature to Give Skin Sparing and Increase Depth Dose with Conventional X-Rays (300 kv.). Edwin J. Liebner, M.D. (by invitation), Chicago, Ill., Walter Moos, Ph.D. (by invitation), Chicago, Ill., Martin Hochhauser, M.D. (by invitation), Chicago, Ill., and Roger Harvey, M.D., Chicago, Ill.
45. Clinical Experience with Roentgen Therapy under High Oxygen Tension. W. B. Seaman, M.D., New York, N. Y., C. S. Sanger, M.D. (by invitation), New York, N. Y., N. DuV. Tapley, M.D. (by invitation), New York, N. Y., H. W. Jacox, M.D., New York, N. Y., and H. L. Atkins, M.D. (by invitation), New York, N. Y.
46. Ten Years' Experience with Low Megavolt Electron Therapy Beam. M. I. Smedal, M.D., Boston, Mass., D. O. Johnston, M.D. (by invitation), Boston, Mass., F. A. Salzman, M.D., Boston, Mass., J. G. Trump, Sc.D., Cambridge, Mass., and K. A. Wright, M.S. (by invitation), Cambridge, Mass.
47. The Use of P^{32} in the Differential Diagnosis of Ocular Neoplasms. C. I. Thomas, M.D. (by invitation), Cleveland, Ohio, J. P. Storaasli, M.D., Cleveland, Ohio, and H. L. Friedell, M.D., Cleveland, Ohio.
48. Newer Trends in Radiotherapy. Justin J. Stein, M.D. (by invitation), Los Angeles, Calif.

NEWS ITEMS

AMERICAN RADIUM SOCIETY ELECTS NEW OFFICERS

At a meeting of the American Radium Society, held at the Broadmoor Hotel in Colorado Springs, Colorado, on May 10–14, 1961, the following officers were elected: President, Robert L. Brown, Atlanta, Ga.; President-Elect, Gilbert H. Fletcher, Houston, Texas; 1st Vice-President, Ralph F. Phillips, New York, N. Y.; 2nd Vice-President, Ethlyn Trapp, Vancouver, B. C., Can.; Secretary, Charles G. Stetson, Englewood, N. J.; and Treasurer, Joseph H. Farrow, New York, N. Y.

The Forty-fourth Annual Meeting of the Society will be held at the Waldorf-Astoria Hotel in New York City on April 1–4, 1962.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY

The twenty-third midsummer radiologic conference of The Rocky Mountain Radiological Society will be held at the Denver Hilton Hotel, Denver, Colorado, August 10, 11, and 12, 1961. The guest speakers will be: Harold G. Jacobson, M.D., Director, Division of Diagnostic Radiology, Montefiore Hospital, 210th Street and Bainbridge Rd., New York 67, N. Y.; Walter T. Murphy, M.D., Director of Therapeutic Radiology, Roswell Park Memorial Institute, Buffalo 3, N. Y.; and William B. Seaman, M.D., Director, Department of Diagnostic Radiology, Columbia-Presbyterian Medical Center, 622 West 168th Street, New York 32, N. Y.

Friday, the 11th, has been dedicated to Dr. Eugene Pendergrass. The Society will honor him for his vast contributions to radiology on the year of his retirement. On this day the program will consist of papers by former residents of Dr. Pendergrass. These speakers will be: Kenneth D. A. Allen, M.D., Denver, Colorado; J. Gershon-Cohen, M.D., Philadelphia, Pennsylvania; Roderick L. Tondreau, M.D., Meadowbrook, Pennsylvania; James C. Katterjohn, M.D., Indianapolis, Indiana; John F. Weigen, M.D., Palo Alto, California; Gilbert W. Heublein, M.D., Hartford, Connecticut; John M. Dennis, M.D., Baltimore, Maryland; C. Richard Perryman, M.D., Pittsburgh, Pennsylvania; Harold Atkins, M.D., New York, New York; Robert P. Barden, M.D., Philadelphia, Pennsylvania; Vern W. Ritter, M.D., Santa Rosa, California; Ellwood W. Godfrey, M.D., Princeton, New Jersey; Nathan P. Salner, M.D., Philadelphia, Pennsylvania; and J. Robert Andrews, M.D., Bethesda, Maryland.

At the annual banquet a plaque will be presented to Dr. Pendergrass in recognition of this occasion. A plaque will also be presented to Dr. Milton Berg, Bismarck, North Dakota, President of the Radiological Society of North America. This will be presented in recognition of his many years of faithful service to the Rocky Mountain Radiological Society and his many contributions to medicine and radiology.

WORLD ASSOCIATION OF ACADEMIC PROFESSORS OF MEDICAL RADIOLOGY

A meeting of the World Association of Academic Professors of Medical Radiology will be held at the Cantonal Hospital, Zürich, Switzerland, from Friday, October 13, until Sunday, October 15, 1961. A banquet will be given on Saturday, October 14, 1961, by the President of the Association, Professor H. R. Schinz, M.D.

Report of intended attendance at the meeting as well as requests for information concerning hotel facilities, etc. should be addressed to the Secretary of the Association, Prof. U. Cocchi, M.D., Kantonsspital, Zürich, no later than June 30, 1961.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

Nuclear Explosions and Their Effects. Revised and enlarged edition. Foreword by Jawaharlal Nehru. Cloth. Price, $4.00. Pp. 340 and 5 charts. The Publications Division, Ministry of Information and Broadcasting, Post Box No. 2011, Old Secretariat, Delhi 8, India, 1958.

In the foreword to the first edition of this book, which was published in June, 1956, Nehru stated: "About a year ago I suggested to the Defence Science Organization of the Government of India that an objective study might be made with the material available of the consequences of the use of nuclear, thermonuclear and other weapons of mass destruction." D. S. Kothari of the Indian Defence Science Organization was entrusted with this task and with the assistance of H. Bhabha, in charge of the atomic energy work, and V. R. Khanolkar, head of the cancer research institute, collected the material upon which this revised and almost completely rewritten edition is based.

After a few historical remarks on the development of nuclear energy, starting with the discovery of x-rays by Röntgen in 1895, the authors describe in detail fission and fusion explosions, fission energy and radioactivity of fission products, and the effects of atomic bombs and of high-yield explosions. This is followed by detailed and valuable presentations of the subjects on long-term radiologic hazards, such as global fallouts and notably of genetic effects. A rather extensive appendix gives further information on fission explosion and critical size, thermonuclear reactions, catalyzed fusion reaction, dose rates from radioactive sources inside and outside the body, and nuclear explosions and the weather.

This book, which also has been published in Germany and Japan, gives an excellent presentation of the dangers of large nuclear bombs. It is written with great clarity in a style that the reader who knows little about physics can well understand. It can be highly recommended.

Otto Glasser, Ph.D.

Nuclear Reactors for Power Generation. Edited by E. Openshaw Taylor, B.Sc., D.I.C., M.I.E.E., M.Am.I.E.E., F.R.S.E., Assistant Professor of Electrical Engineering, Heriot-Watt College, Edinburgh, Scotland. Cloth. Price, $7.50. Pp. 144, with 51 illustrations. Philosophical Library Inc., 15 East Fortieth Street, New York 16, N. Y., 1958.

This book is based on a series of advanced lectures delivered at the Heriot-Watt College in Edinburgh, Scotland, by the editor, E. O. Taylor, with the assistance of J. H. Bowen, W. H. J. Childs, J. M. Kay, W. L. Kidd, and A. B. McIntosh. After introductory remarks about the world energy requirements and present fuel reserves, the nuclear energy programs of America, Great Britain, Russia and some smaller European countries are discussed. This is followed by chapters on fundamental nuclear physics, basic design and various types of nuclear reactors, building materials used in reactors, safety and instrumentation and the economics of different types of reactors.

The book is well written and printed and the numerous line drawings are very helpful in interpreting the text. It should be read by all those who are interested in atomic reactors, whether they are used for the production of power or of isotopes.

Otto Glasser, Ph.D.

Le Radiocalcium dans l'Étude des Os. By R. Ponlot, Chercheur agréé de l'Institut Interuniversitaire des Sciences Nucléaires, Chef de Travaux à l'Université de Louvain. Paper. Pp. 254, with 86 illustrations. Editions Arscia S.A., 60, rue de l'Etuve, Bruxelles, Belgium; Masson et Cie, 120 Boulevard Saint-Germain, Paris 6e, France, 1960.

In 1955, Dr. J. Vincent, in a monograph titled "Experiments on the Constitution of the Adult Bone," had demonstrated that, in order to obtain good interpretation of a contact autoradiograph of the osseous tissue, it is necessary to compare it not only with the histoiogic section but also with the microradiograph of the bone. Because of this, the interest has been focussed on the mineral portion of the bone entailing the utilization of Ca^{45} isotope studies.

Ponlot has pursued such studies since 1955,

performing numerous investigations, mainly on the physiopathology of the bone; *i.e.*, the role of the osseous skeleton in the general metabolism of calcium in the system. In the present excellent monograph he expounds the results of his Ca^{45} investigations with regard to the histology of bone aiming to establish the morphologic significance of autoradiographs performed *in vivo*.

The first five chapters deal with the histologic pattern of the compact adult bone and the structural changes of the haversian system. The affinity of collagen for mineral salts, revealed by certain experiments *in vitro*, is probably not the only factor determining the calcification *in vivo*.

The next five chapters discuss autoradiographic studies of the adult compact bone made 7 hours, 12 days, and 6 weeks following injection of Ca^{45}, and an attempt is made to interpret the phenomena of calcification and mineral salt exchanges. The remaining ten chapters are devoted to detailed investigations of the bone growth particularly of the tibia and vertebra.

The author describes *in extenso* the numerous investigations performed and uses many pertinent illustrations of the finest quality to emphasize the important points of the discussion. He concludes by defining the possible applications both for experimental and clinical investigations in Orthopedic Surgery and the role which Sr^{90} and Ca^{45} respectively may occupy in these investigations.

Jacques Lespérance, M.D.

BOOKS RECEIVED

Roentgenology of Intracranial Meningiomas. By Sidney P. Traub, M.D., Associate Professor of Radiology, University of Saskatchewan College of Medicine; Assistant Director of Radiology, University Hospital, Saskatoon, Canada; Honorary Consultant Radiologist, Saskatchewan Cancer Commission; and formerly, Fellow in Neuroradiology, Montreal Neurological Institute, Montreal, Canada. Cloth. Price, $14.00. Pp. 238, with 128 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

Diseases of the Intervertebral Disc and Its Surrounding Tissues. By Reuben Rabinovitch, B.A., M.Sc., M.D., Assistant Professor in Neurology and Neurosurgery, McGill University, Faculty of Medicine, Montreal; and Assistant Neurologist, The Department of Neurology and Neurosurgery, The Royal Victoria Hospital and The Montreal Neurological Institute. Cloth. Price, $8.50. Pp. 152, with 78 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

Radioactivity in Man; A Symposium Held at the Vanderbilt University School of Medicine. Edited by George R. Meneely, M.D. Cloth. Price, $16.50. Pp. 491, with numerous illustrations and tables. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

The Pathology of Ionizing Radiation. By Shields Warren, M.D., Sc.D., LL.D., Professor of Pathology, Harvard Medical School at the New England Deaconess Hospital; Pathologist, New England Deaconess and New England Baptist Hospitals, Boston, Mass. Cloth. Price, $3.00. Pp. 42, with 17 illustrations. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill., 1961.

A Manual of Cutaneous Medicine. By Donald M. Pillsbury, M.A., D.Sc. (Hon.), M.D., F.A.C.P., Professor and Chairman of the Department of Dermatology, University of Pennsylvania School of Medicine; Director, Commission on Cutaneous Diseases, Armed Forces Epidemiological Board; and President, XII International Congress of Dermatology; Walter B. Shelley, M.D., Ph.D., F.A.C.P., Professor of Dermatology, University of Pennsylvania School of Medicine; and National Consultant in Dermatology to the Surgeon General, U. S. Army and U. S. Air Force; and Albert M. Kligman, M.D., Ph.D., Professor of Dermatology, University of Pennsylvania School of Medicine; and Professor of Dermatology, University of Pennsylvania Graduate School of Medicine. Cloth. Pp. 430, with 234 illustrations. W. B. Saunders Company, 218 W. Washington Square, Philadelphia 5, Pa., 1961.

Das Carcinoma Colli Uteri, Seine Prognose und Behandlung; mit Besonderer Berücksichtigung der Strahlentherapie. By Dr. med. Wilhelmine Janisch-Raskovic, ehe. Strahlentherapeutin der vorm. Staatl. Frauenklinik Chemnitz jetzt Städt. Frauenklinik Karl-Marx-Stadt. Cloth. Pp. 649, with many illustrations and tables. Georg Thieme Verlag, Herdweg 63, Stuttgart, Germany, 1961.

Medical Almanac 1961–62. Compiled by Peter S. Nagan, A.B., M.A., M.S. Paper. Pp. 528. W. B. Saunders Company, 218 W. Washington Square, Philadelphia 5, Pa., 1961.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. C. Allen Good, Mayo Clinic, Rochester, Minn. Annual meeting: Deauville Hotel, Miami Beach, Fla., Sept. 26–29, 1961.

American Radium Society
Secretary, Dr. Charles G. Stetson, 350 Engle Street, Englewood, N. J. Annual meeting: Waldorf-Astoria Hotel, New York, N. Y., April 1–4, 1962.

Radiological Society of North America
Secretary, Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb.
Treasurer, Dwight Vincent Needham, 713 E. Genessee St., Syracuse, N. Y. Annual Meeting: Palmer House, Chicago, Ill., Nov. 26–Dec. 1, 1961.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Illinois. Annual meeting: Roosevelt Hotel, New York, N. Y. Feb. 7–10, 1962.

Section on Radiology, American Medical Association
Secretary, Dr. Clyde A. Stevenson, Sacred Heart Hospital, West 101 Eighth Ave., Spokane 4, Wash. Annual meeting: New York City, June 26–30, 1961.

American Board of Radiology
Secretary, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building. Rochester, Minn. The Fall 1961 examination will be held at the Shoreham Hotel, Washington, D. C., December 4–7, inclusive; the deadline for filing applications is July 1, 1961. A Special Examination in Nuclear Medicine will be offered if there are sufficient applications.

American Association of Physicists in Medicine
Secretary-Treasurer, Charles S. Simons, University of Michigan Hospital, Ann Arbor, Mich. Annual meeting to be announced.

Tenth International Congress of Radiology
Secretary-General, Dr. Carleton B. Peirce, Royal Victoria Hospital, Montreal 2, Quebec, Canada. Meets in Montreal, Aug. 26–Sept. 1, 1962.

Seventh Inter-American Congress of Radiology
Counselor for the United States, Dr. J. A. del Regato, Penrose Cancer Hospital, 2200 North Cascade Avenue, Colorado Springs, Colorado. The meeting will be held in São Paulo, Brazil, September 3–10, 1961.
Secretary-General, Dr. Walter Bomfim-Pontes, Rua Cesario Motta, No. 112, São Paulo.

Alabama Radiological Society
Secretary, Dr. J. A. Meadows, Jr., Medical Arts Bldg., Birmingham 5, Ala. Meets time and place Alabama State Medical Association.

American Nuclear Society
Executive-Secretary, Octave J. Du Temple, 86 E. Randolph St., Chicago, Ill.

Arizona Radiological Society
Secretary, Dr. Don E. Matthieson, 926 East McDowell Rd., Phoenix, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Radiological Society
Secretary, Dr. J. B. Scruggs, Arkansas Baptist Hospital, Little Rock, Ark. Meets every three months and also at time and place of State Medical Association.

Association of University Radiologists
Secretary, Dr. Melvin M. Figley, Department of Radiology, University of Washington, Seattle 5, Wash. Annual meeting to be announced.

Atlanta Radiological Society
Secretary, Dr. Wilson T. Edenfield, 35 Linden Ave., N.E., Atlanta 8, Ga. Meets monthly, except during three summer months, on second Friday evening.

Blockly Radiological Society
Secretary, Dr. Samuel Finkelman, 101 S. Twentieth St., Philadelphia, Pa.

Brooklyn Radiological Society
Secretary, Dr. Joseph Arcomano, 168 Clinton St., Brooklyn 1, N. Y. Meets first Thursday of each month October through May.

Buffalo Radiological Society
Secretary, Dr. Kenneth H. Seagrave, 537 Delaware Ave., Buffalo 2, N. Y. Meets second Monday evening each month, October to May inclusive.

Central New York Radiological Society
Secretary, Dr. Joseph A. Head, 150 Marshall St., Syracuse, N. Y. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary, Dr. Robert L. Freidman, Grant Hospital, Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, November, January, March and May at Fort Hayes Hotel, Columbus, Ohio.

Central Society of Nuclear Medicine
Secretary, Dr. Robert S. Landauer, Radiation Center Building, 1903 West Harrison St., Chicago 12, Ill.

Chicago Roentgen Society
Secretary, Dr. William F. Hutson, 5145 N. California Ave., Chicago, Ill. Meets second Thursday of each month, October to April except December at the Sheraton Hotel at 8:00 P.M.

Cleveland Radiological Society
Secretary, Dr. Norman E. Berman, 14404 S. Park Blvd., Shaker Hgts. 20, Ohio. Meetings at 7:00 P.M. on fourth Monday of each month from October to April at Tudor Arms Hotel.

Colorado Radiological Society
Secretary, Dr. Seward Imes, 1845 High St., Denver, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. James L. Krieger, 85 Jefferson St., Hartford, Conn. Meets first Friday in February and April.

Dallas-Fort Worth Radiological Society
Secretary, Dr. F. J. Bonte, 5201 Harry Hines Blvd., Dallas 35, Texas. Meets monthly, third Monday, at Greater Fort Worth International Airport at 6:30 P.M.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. Kenneth L. Krabbenhoft, Harper Hospital, Detroit 1, Mich. Meets monthly first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

East Tennessee Radiological Society
Secretary, Dr. J. Marsh Frere, Jr., 205 Medica Arts Building, Knoxville, Tenn. Meets in January and September.

Eastern Conference of Radiology
Secretary, Arrangements Committee, Dr. Philip Myers, Baltimore City Hospital, Baltimore 24, Md. Annual meeting to be announced.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

EASTERN RADIOLOGICAL SOCIETY
Secretary, Dr. James F. Martin, North Carolina Baptist Hospital, Winston-Salem, N. C. Meets at Mid Pines Club, Southern Pines, N. C., April 29–May 2, 1962.

FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. Alfred G. Levin, 837 DuPont Bldg., Miami, Fla. Meets twice annually, in the spring with the annual State Society Meeting, and in the fall.

FLORIDA WEST COAST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joseph C. Rush, 1800 Druid Rd., Clearwater, Fla.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. George W. Brown, Griffin, Ga. Meets in spring and fall with Annual State Society Meeting.

GREATER MIAMI RADIOLOGICAL SOCIETY
Secretary, Dr. Carl E. Balli, 907–8 Huntington Medical Building, Miami 32, Fla. Meets monthly third Wednesday at 8:00 P.M. at Jackson Memorial Hospital, Miami, Fla.

GREATER ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary, Dr. William E. Powers, St. Louis, Mo.

HOUSTON RADIOLOGICAL SOCIETY
Secretary, Dr. John Douglas Reeve, Texas Medical Center Library, Jesse H. Jones Library Bldg., Houston 25, Texas. Meets last Monday each month, Seminar Room, Doctors' Club of Houston.

IDAHO STATE RADIOLOGICAL SOCIETY
Secretary, Dr. George H. Harris, Bannock Memorial Hospital, Pocatello, Idaho. Meets in the Spring and Fall.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. George A. Miller, Carle Hospital Clinic, Urbana, Ill. Meets in spring and fall.

INDIANA ROENTGEN SOCIETY, INC.
Secretary, Dr. David E. Wheeler, 1500 North Ritter, Indianapolis, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

IOWA RADIOLOGICAL SOCIETY
Secretary, Dr. L. L. Maher, 1419 Woodland Ave., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

KANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. Roger K. Wallace, Riley County Hospital, Manhattan, Kansas. Meets in spring with State Medical Society, and in winter on call.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. Lawrence A. Davis, 226 East Chestnut St., Louisville, Ky. Meets monthly on second Friday at Seelbach Hotel, Louisville.

KINGS COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Abraham Berens, 1917 Bedford Ave., Brooklyn 25, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

LOS ANGELES RADIOLOGICAL SOCIETY
Secretary, Dr. Walter Stilson, 1720 Brooklyn Ave., Los Angeles, Calif. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles.

MAINE RADIOLOGICAL SOCIETY
Secretary, Dr. Albert A. Poulin, Thayer Hospital, Waterville, Maine. Meets in June, September, December and April.

MARYLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Nathan B. Hyman, 1805 Eutaw Place, Baltimore 17, Md.

MEMPHIS ROENTGEN SOCIETY
Secretary, Dr. Hollis H. Halford, Kennedy V.A. Hospital, Department of Radiology, Memphis 15, Tenn. Meets first Monday of each month at John Gaston Hospital.

MIAMI VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. William D. Roberts, 2197 Los Arrow Dr., Dayton 9, Ohio. Meets second Friday of fall and winter months.

MID-HUDSON RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph Sorrentino, St. Francis Hospital, Poughkeepsie, N. Y. Meets 8:30 P.M., fourth Wednesday each month, September to May.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. Joseph F. Wepfer, 5000 W. Chambers St., Milwaukee 10, Wis. Meets monthly on fourth Monday, October through May, at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. Donald H. Peterson, 853 Medical Arts Bldg., Minneapolis 2, Minn. Meets three times annually, in fall, winter and spring.

MISSISSIPPI RADIOLOGICAL SOCIETY
Secretary, Dr. Jack K. Goodrich, University Medical Center, Jackson, Miss. Meets third Thursday of each month at Hotel Edwards, Jackson, at 6:00 P.M.

MONTANA RADIOLOGICAL SOCIETY
Secretary, Dr. J. K. Boughn, 35 11th Ave., Helena, Montana. Meets at least once a year.

NASSAU RADIOLOGICAL SOCIETY
Secretary, Dr. Alan E. Baum, 100 Nowbridge Rd., Hicksville, N. Y. Meets second Tuesday of the month in February, April, June, October and December.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. Ronald E. Waggener, The Radiologic Center, Nebraska Methodist Hospital, Omaha 31, Nebraska. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Robert E. Wise, 605 Commonwealth Ave., Boston 15, Mass. Meets third Friday of each month, October through May at The Longwood Towers, Brookline, Mass.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY
Secretary, Dr. Paul Y. Hasserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. Bernard Wolf, Mt. Sinai Hospital, New York, N. Y. Meets monthly on third Monday, New York Academy of Medicine at 4:30 P.M.

NORTH CAROLINA RADIOLOGICAL SOCIETY
Secretary, Dr. A. B. Croom, 624 Quaker Lane, High Point, N. C. Meets in the spring and fall each year.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. R. F. Raasch, Post Office Box 990, Dickinson, North Dakota. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

NORTH FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. Charles H. Newell, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.

NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Lester I. Citrin, St. Mary's Hospital, Troy, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.

NORTHERN CALIFORNIA RADIOLOGICAL SOCIETY
Secretary, Dr. Rob H. Kirkpatrick, 1219 28th St., Sacramento, Calif. Meets at dinner last Monday of each month, September to June.

OHIO STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Paul D. Meyer, 125 S. Grant Ave., Columbus, Ohio. Annual meeting to be announced.

OKLAHOMA STATE RADIOLOGICAL SOCIETY
Secretary, Dr. E. D Greenberger, Medical Arts Bldg., McAlester, Okla. Meets in January, May and October.

OREGON RADIOLOGICAL SOCIETY
Secretary, Dr. George R. Satterwhite, 1123 S.W. Yamhill, Portland, Ore. Meets monthly from October to June on

the second Wednesday of each month at 8:00 P.M. at the University Club.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. John N. Burkey, 555 Dental Bldg., Seattle, Wash. Annual meeting to be announced.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco 8, Calif. Meets annually during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. Frederick R. Gilmore, 234 State St., Harrisburg, Pa. Annual meeting to be announced.

Philadelphia Roentgen Ray Society
Secretary, Dr. Robert B. Funch, Department of Radiology, Germantown Hospital, Philadelphia 44, Pa. Meets first Thursday of each month, at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Ross H. Smith, St. Margaret Memorial Hospital, Forty-Sixth St., Pittsburgh 1, Pa. Meets second Wednesday of month, October through June at Park Schenely Restaurant.

Radiological Section, Baltimore Medical Society
Secretary, Dr. James K. V. Willson, 1100 N. Charles St., Baltimore 1, Md. Meets third Tuesday each month, September to May, inclusive.

Radiological Society of Greater Cincinnati
Secretary, Dr. Donald Janny, Cincinnati, Ohio. Meets monthly from September to May on first Monday of each month at 7:30 P.M. at the Cincinnati General Hospital.

Radiological Society of Hawaii
Secretary, Dr. Philip S. Arthur, 274 Young Hotel Bldg., Honolulu, Hawaii. Meets third Monday of each month at 7:30 P.M.

Radiological Society of Greater Kansas City
Secretary, Dr. J. Stewart Whitmore, 1010 Rialto Bldg., Kansas City, Mo. Meets last Friday of each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana
Secretary, Dr. Robyn Hardy, 4324 Magnolia St., New Orleans 15, La. Meets annually during Louisiana State Medical Society meeting.

Radiological Society of New Jersey
Secretary, Dr. Austin J. Tidaback, 912 Prospect Ave., Plainfield, N. J. Meets at Atlantic City at time of State Medical Society meeting and in November in Newark, N. J.

Radiological Society of New York State
Secretary-Treasurer, Dr Mario C. Gian, 610 Niagara St., Buffalo 1, N. Y. Annual meeting to be announced.

Radiological Society of South Dakota
Secretary-Treasurer, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.

Radiological Society of Southern California
Secretary, Dr. Joseph F. Linsman, 436 N. Roxbury Dr., Beverly Hills, Calif.

Redwood Empire Radiological Society
Secretary, Dr. Lee E. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Robert H. Greenlaw, 260 Crittenden Blvd., Rochester 20, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. John H. Freed, 4200 East Ninth Ave., Denver 20, Colo. Annual meeting: Denver Hilton Hotel, Denver, Colo., Aug. 10–12, 1961.

San Antonio-Military Radiological Society
Secretary, Dr. Hugo F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Texas. Meets third Wednesday each month in Fort Sam Houston Officer's Club at 6:30 P.M.

San Diego Radiological Society
Secretary, Dr. Stanley A. Moore, 2466 First Ave., San Diego 1, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. M. A. Sisson, 450 Sutter St., San Francisco 8, Calif. Meets quarterly at the San Francisco Medical Society, 250 Masonic Ave., San Francisco 18, Calif.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Connecticut State Medical Society
Secretary, Dr. Wayne P. Whitcomb, Hospital of St. Raphael, New Haven, Conn. Meetings are held bimonthly.

Section on Radiology, Medical Society of the District of Columbia
Secretary, Dr. William E. Sheely, 1746 K St., N.W., Washington 6, D. C. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. William Meszaros, 1825 W. Harrison St., Chicago, Ill.

Section on Radiology, Southern Medical Association.
Secretary. Dr. Seymour Ochsner, Ochsner Clinic, 3503 Prytania St., New Orleans 15, La. Annual meeting to be announced.

Shreveport Radiological Club
Secretary, W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. Richard G. Lester, 412 Union St., S.E., Minneapolis 14, Minn. Annual meeting: Deaville Hotel, Miami Beach, Fla., Sept. 25, 1961.

Society of Nuclear Medicine
Secretary, Dr. Robert W. Lackey, 452 Metropolitan Bldg., Denver 2, Colo. *Administrator*, Samuel N. Turiel, 430 N. Michigan Ave., Chicago 11, Ill. Annual meeting to be announced.

South Bay Radiological Society
Secretary, Dr. Stanford B. Rossiter, 1111 University Dr., Menlo Park, Calif. Meets second Wednesday of each month.

South Carolina Radiological Society
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

Southern Radiological Conference
Secretary, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 4097, Mobile, Ala.

Southwestern Radiological Society
Secretary, Dr. Ralph S. Clayton, 1501 Arizona, Bldg. 2-A, El Paso, Texas. Meets second Tuesday of each month.

Tennessee Radiological Society
Secretary, Dr. B. M. Brady, St. Joseph Hospital, Memphis, Tenn. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 402 Professional Bldg., 1216 Pennsylvania Ave., Fort Worth 4, Texas. Annual meeting: Austin, Texas, Jan. 19 and 20, 1962.

Tri-State Radiological Society
Secretary, Dr. John Marchand, 118 S.E. First St., Evansville, Ind. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M. at Elks Club in Evansville.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society
Secretary, Dr. Richard Y. Card, St. Mark's Hospital, Salt Lake City, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Virginia Radiological Society
Secretary, Dr. Powell G. Dillard, Jr., 715 Church Street, Lynchburg, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Joseph T. Houk, 14303 Ambaum Blvd., Seattle 66, Wash. Meets third Monday of each month from September through April at the University of Washington Medical School.

West Virginia Radiological Society
Secretary, Dr. Karl J. Myers, 112 N. Woods St., Philippi, W. Va. Meets concurrently with Annual Meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester Radiological Society
Secretary, Dr. Anthony A. Maglione, Westchester Academy of Medicine, Section on Radiology, Purchase, N. Y. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society
Secretary, Dr. Howard G. Bayley, 116 Iroquois Parkway, Beaver Dam, Wis. Annual meeting each spring in various places.

Cuba, Mexico, Puerto Rico and Central America

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
Secretary-General, Dr. Julio Toriello, 11 Calle 2-37, zona 1, Guatemala. Meets annually in a rotating manner in the six countries.

Sociedad de Radiología de El Salvador
Secretary, Dr. Rafael Vega Gómez.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad de Radiología y Fisioterapía Cubana
Secretary, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.

Sociedad Costarricense de Radiologia
Secretary, Dr. James Fernández Carballo, Apartado VIII, San José, Costa Rica.

Sociedad Mexicana de Radiología, A. C.
Calle del Oro No. 15, México 7, D. F
Secretary-General, Dr. E. Alvarez Hernández. Meets first Monday of each month.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital, chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. César E. Rosa-Perez, Fondo del Segura del Estado, Parada 1, San Juan 8, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Secretary, Dr. Odilon Raymond, 5400 Blvd. Gouin, Quest, Montreal, Que. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. R. D. Hoare, 32 Welbeck St., London, W. 1. Meets monthly from October until May.

Edmonton and District Radiological Society
Secretary, Dr. S. C. Windle, 105 Northgate Bldg., Edmonton, Alberta. Meets first Tuesday of each month, Oct. to May.

Faculty of Radiologists
Honorary Secretary, Dr. R. A. Kemp Harper, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting to be announced.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1.

Canadian Association of Radiologists
Honorary Secretary, Dr. Robert G. Fraser, *Associate Honorary Secretary*, Dr. Jean-Louis Léger, 1555 Summerhill Ave., Montreal 25, Que. Annual meeting: Marlborough Hotel, Winnipeg, Man., Jan. 22–24, 1962.

Montreal Radiological Study Club
Secretary, Dr. F. McConnell, 1650 Cedar Ave., Montreal, Quebec. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française d'Electro-Radiologie Médicale
General Secretary, Dr. Maurice Dufresne, 1560 Sherbrooke (East), Montreal, Canada. Meets third Saturday each month.

Toronto Radiological Society
Secretary, Dr. L. R. Harnick, Toronto Western Hospital, 399 Bathurst St., Toronto, Ontario. Meets second Monday of each month September through May.

College of Radiologists of Australia
Honorary Secretary, Dr. E. A. Booth, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Victor A. Añaños, Instituto de Radiologia, Santa Fe 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional del Centenario, Santa Fe 1300, Rosario.

Colégio Brasileiro de Radiologia
Secretary-General, D. Camillo Segreto, Avenida Angélica, 1.170, Caixa Postal 5984, São, Paulo, Brazil.

Sociedad Argentina de Radiología, Junta Central, Buenos Aires
Secretary, Dr. Edgardo O. Olcese, Santa Fé 1171, Buenos Aires. Meetings are held monthly.

Sociedad Bolivana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1596, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. J. P. Velasco, Avenida Santa María 0410, Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary, Dr. Alberto Mejía Diazgranados, Carrera 13, No. 25–31, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología y Fisioterapía
Secretary, Dr. Publio Vargas P., Casilla 1242, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary, Dr. Luis Pinillos Ganoza, Apartado 2306, Lima, Perú. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta 218, Lima.

Sociedad de Radiologia del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Ernesto H. Cibils, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiologia de Pernambuco
Secretary, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenologia y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Carlos A. Oulton, Santa Rosa 447, Córdoba, Argentina.

Sociedad Venezolana de Radiología
Secretary-General, Dr. Rubén Merenfeld, Apartado No. 9362, Candelaria, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 256 Chaussée de Wavre, Heverlee-lez-Louvain, Belgium. Meets in February, March, May, June, October, November and December.

Société Française d'Electroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris.
Secretary-General, Dr. Ch. Proux, 9, rue Daru, Paris 8e, France.

Československá Společnost pro Roentgenologii a Radiologii
Secretary, Dr. Robert Poch, Praha 12, Šrobárova 50, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Deutsche Röntgengesellschaft
Secretary, Professor Dr. med. H. Lossen, Universitäts-Röntgeninstitut. Lagenbeckstr. 1, Mainz, Germany. Annual meeting to be announced.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano, Torino, Italy. Meets annually.

Nederlandse Vereniging voor Electrologie en Röntgenologie
Secretary, Dr. J. R. von Ronnen, Violenweg 14, den Haag. Netherlands.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Española de Radiología y Electrología Médicas y Medicina Nuclear
Secretary, Dr. D. Aureo Gutierrez Churruca, Esparteros, No. 9, Madrid, Spain. Meets monthly in Madrid.

Schweizerische Gesellschaft für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

India

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

HEAD

BRANNON, EARL W. Congenital malformation of the atlanto-axial joint with dislocation. *J. Bone & Joint Surg.*, Dec., 1960, *42A*, 1377–1380. (From: Department of Orthopaedic Surgery, United States Air Force Hospital, Lackland, San Antonio, Texas.)

The author discusses the unique anatomy and function of the first two cervical segments. The causes of dislocation at this level, with emphasis on the effects of dysplasia of the dens, are reviewed. In a case report he describes the clinical findings and treatment.

The annular atlas, supporting the head and surrounding the odontoid process, articulates with the axis in a manner which permits rotation at the atlantoaxial joint. The efficiency of the transverse and check ligaments in maintaining stability is dependent upon the integrity and normal development of the osseous elements at this level; because of the intimate structural relationship to the spinal cord, minor luxations may produce neurologic abnormalities.

Dislocation may result: (1) when the anterior or posterior arch of the atlas is fractured; (2) with fracture of the odontoid process; (3) as a result of demineralization of bone with resultant disruption of ligamentous attachments, as might be caused by infection; and (4) due to congenital absence or hypoplasia of the odontoid process. With hypoplasia the defect may be subclinical but the excessive mobility which is produced tends to become progressive, with weakening of other ligamentous attachments and the eventual appearance of neurologic signs.

When instability is the result of hypoplasia of the odontoid process, symptoms may first be noted in the third decade. They consist of pain in the neck, shoulders and occipital region, which is aggravated by lifting, and there may be associated numbness and paresthesia of the arms. Physical signs may consist of some limitation of neck extension and induction of the pain pattern when the neck is flexed. The spinous process of the axis is prominent. Deep tendon reflexes in all extremities may be hyperactive.

In the case cited, roentgenograms showed a malformed and shortened odontoid process with forward dislocation of the occiput and atlas; dislocation was exaggerated upon flexion. Spine fusion was performed from the occiput to the fifth cervical vertebra. At operation, partial absence of the laminae, a bifid axis and a lack of development of the ligaments overlying the dura were demonstrated. The surgical treatment was successful.—*Jack Reynolds, M.D.*

LUCCARELLI, S., and DE FERRARI, U. Studio clinico-radiologico di un caso di origine anomala (dall'a. carotide esterna) dell'arteria vertebrale sinistra. (Clinical and radiologic study of a case of anomalous origin [from the external carotid artery] of the left vertebral artery.) *Radiol. med.*, Oct., 1960, *46*, 963–974. (From: Divisione di Neuro-Psicopatologia and Istituto di Radiologia, Ospedale Maggiore di Milano, Milano, Italy.)

The anomaly described is one of origin and of course of the left vertebral artery. It was casually discovered in an angiographic examination. The presence of such a singular vascular malformation was not suspected clinically.

The angiographic examination was carried out as follows: The contrast medium was injected into the left carotid artery at the level of the common trunk. The internal carotid artery was visualized normally and, at the same time, to the authors' surprise, vessels of the vertebral basilar system were opacified.

It was determined surgically and roentgenologically that the left external carotid, after having given origin to the superior thyroid, lingual and maxillary arteries, continued cephalad, assuming the morphologic and functional characteristics of the vertebral artery and coursing totally outside the canal formed by the transverse foramina.

The authors give an excellent summary of the embryology and anatomy of the anomalies of the vertebral artery.

Clinically, their patient, a hypertensive female fifty-one years of age, presented the picture attributed to softening of the posterior sylvian portion of the left cerebral hemisphere.—*Anthony A. Blasi, M.D.*

ECKER, ARTHUR, and PERL, THEODORE. Percutaneous injection of the thalamus in Parkinsonism; a preliminary report: relief of bilateral facial grimaces. *A.M.A. Arch. Neurol.*, Sept., 1960, *3*, 271–278. (From: Departments of Surgery [Neurosurgery] and of Radiology, State University of New York, Upstate Medical Center, Syracuse, N. Y.)

Injection of the thalamus using the well-known cannula and balloon technique results in a mortality of 2.4 per cent and an additional complication rate, usually hemiplegia, of 3 per cent. With this new method, the target in the ventrolateral nucleus of the thalamus is determined by measuring from the calcified pineal body. If it is not calcified, a pneumoencephalogram is required for localization of the pineal body, the target is aligned through the center of the foramen ovale to the skin, thus indicating the point of entry. A 21 gauge guide needle is inserted through the skin to the foramen ovale. Then a longer 26 gauge needle is passed a measured distance through the guide needle to the target, after which 0.2 cc. of 1 per cent procaine or lidocaine is injected through the inner needle to determine the clinical

effect. Finally, small amounts of absolute alcohol (0.1 to 0.2 cc.) are injected to obtain a permanent effect.

The target area is the most anteroinferolateral portion of the thalamus. The target lies 16 mm. lateral to the midline, and 10 mm. in front of the posterior commissure in a horizontal plane 2 mm. above that which includes the anterior and posterior commissures. There is usually dramatic relief of rigidity and/or tremor within seconds. If the symptoms recur after a few minutes, the injection is repeated. When the maximum benefit is obtained, the procedure is concluded by withdrawing the needle.

Seven patients were injected using this method. In all cases contralateral rigidity and tremor were largely or completely relieved.—*Edwin F. Koch, Jr., M.D.*

Neck and Chest

Cathcart, Richard T., Theodos, Peter A., and Fraimow, William. Anthracosilicosis; selected aspects related to the evaluation of disability, cavitation, and the unusual x-ray. *A.M.A. Arch. Int. Med.*, Sept., 1960, *106*, 368–377. (From: Barton Memorial Division, Jefferson Medical College Hospital, Philadelphia, Pa.)

The authors present their findings in regard to selected aspects of the pneumoconiosis problem in anthracite coal workers based on a ten year study of some 1,690 long-term workers.

They found that the degree of anthracosilicosis demonstrated roentgenographically did not necessarily parallel ventilatory function. Ventilatory function and, therefore, the degree of disability appeared to be much more closely related to the degree of concomitant emphysema.

It is well known that tuberculosis is a not infrequent complication of anthracosilicosis and that it is a frequent cause of cavitary disease. None the less, cavitary disease without tuberculosis does occur in anthracosilicosis and this possibility should be recognized. Of 182 miners with cavitary disease, in 18 per cent it was due not to tuberculosis but to ischemic necrosis within conglomerate masses of nodules.

The authors present cases which illustrate the following clinical and roentgen features of ischemic necrosis in anthracosilicosis: (1) cavitation in association with areas of conglomerate fibrosis; (2) sudden and rapid development of a cavity due to liquefaction of a conglomerate area; (3) copious amounts of black sputum; (4) an aseptic clinical course unaccompanied by chills, fever, or leukocytosis; and (5) the fact that this entity does not prognostically influence the course of the underlying anthracosilicosis.

The usual case of anthracosilicosis presents no difficulty in diagnosis. There are numerous cases, however, where the exposure is real and disability is claimed but the roentgen examination does not show the typical silicotic nodules. The roentgen changes may be only increased lung markings and linear fibrosis, mimicking changes frequently seen in men of this age group who have no specific industrial exposure. In such cases lung biopsy is indicated. This procedure has been used in 30 cases and has been most helpful in establishing the presence or absence of silicosis. In 4 of 19 miners with negative roentgenograms, silicotic nodules were found. In 5 of 11 miners with the "linear fibrosis" pattern, evidence of silicosis was found.—*Martin A. Thomas, Major, USAF MC*

MacFadyen, D. J. Wegener's granulomatosis with discrete lung lesions and peripheral neuritis. *Canad. M.A.J.*, Oct., 1960, *83*, 760–764. (From: Department of Medicine, University Hospital, Saskatoon, Saskatchewan, Canada.)

Wegener's granulomatosis comes within that spectrum of disease states ranging from necrotizing and granulomatous processes without vasculitis to vasculitis without granulomata. The granulomatous processes are represented by such diseases as midline lethal granuloma, wherein generalized vascular lesions are usually absent. The group of vasculitis processes without granulomata includes hypersensitivity angiitis.

Within these two extremes lies Wegener's granulomatosis, along with other mixed forms such as allergic angiitis with granulomatosis. Wegener's granulomatosis is distinguished from other members of this group by the predominance of respiratory symptoms and lesions.

The author presents an interesting case of Wegener's granulomatosis. This case featured well-circumscribed granulomata which were similar roentgenographically to metastatic lesions. (One series of such cases demonstrated dense, circular or oval opacities in the lungs in slightly more than 50 per cent of the patients.) The case presented also showed a predominance of vasculitis symptoms but a paucity of respiratory symptoms. The peripheral neuritis which was present closely resembled that found in cases of periarteritis nodosa.—*Charles H. Helmen, M.D.*

Melot, G., and Bollaert, A. Tentative de mesure de la circulation pulmonaire par l'angiopneumographie. (An attempt to measure pulmonary circulation by angiopneumography.) *J. belge de radiol.*, May, 1960, *43*, 445–478. (From: Service de Radiodiagnostic de l'Hôpital universitaire Saint-Pierre, Bruxelles, Belgium.)

In order to understand the pulmonary circulatory function, it is necessary to study the extent of the pulmonary vascular bed, the caliber of the pulmo-

nary vessels and the rate of intrapulmonary circulation. This report is based on angiopneumographies safely performed on 450 patients with various pulmonary diseases. The results were supported by bronchospirometric findings in most cases.

Studies of the caliber of the vessels established the following normal values, which are comparable to those reported by Dotter and Steinberg: pulmonary artery, 32 mm.; left branch, 20 mm.; right branch, 22 mm.; lobar artery, 12 mm.; segmental artery, 7.5 mm.; subsegmental artery, 6 mm.; peripheral, 1–2 mm.; pulmonary veins, 12 mm.

Circulation time, as determined by the time needed for opacification of the pulmonary artery and its corresponding vein, is 2–3 seconds. The duration of the arterial, capillary and venous phases of opacification is 3 seconds with a maximum of $4\frac{1}{2}$ seconds.

The authors report in detail 7 cases illustrating striking pathologic findings. Case I: long-standing tuberculosis of the right upper lobe with accompanying collapse, hydrothorax, and retraction of the heart and mediastinum to the left. Angiopneumography revealed markedly increased vascularity of the right lung with some increase in caliber, despite retraction of the pulmonary artery to the left. There was nonvascularization of the left lung. Case II: a history of left pneumonectomy for bronchogenic carcinoma. Angiopneumography demonstrated marked hypervascularization of the right lung. Case III: thoracoplasty with resection of the six left upper ribs and accompanying lung collapse. Angiopneumography revealed decreased vascularity of the left lung with well-developed right pulmonary vascularization. Case IV: left hilar carcinoma and left upper lobe atelectasis. Angiopneumography showed decreased aeration and decreased vascularization of the left lung but normal vascularization of the right lung. Case V: a history of left hydropneumothorax with almost complete collapse of the left lung and marked residual pleural thickening and retraction of the heart and mediastinum to the left. Angiopneumography visualized markedly decreased vascularity of the left lung and normal vascularity in the right lung. Case VI: carcinoma of the right main bronchus, collapse of the right lung, and retraction of the heart and mediastinum to the right. There was no ventilation of the right lung and markedly decreased vascularity was demonstrated by angiopneumography; the vascular bed on the left side was normal. Case VII: generalized pulmonary emphysema of long standing. Angiopneumography revealed markedly decreased vascularity of the pulmonary bed with decreased caliber of the vessels; however, there was dilatation of the proximal or larger vessels, bilaterally.

The authors find that in significant unilateral lesions (tuberculosis, cancer, pyosclerosis) there is a progressive decrease in the caliber of the arteries and veins on the affected side, whereas on the healthy or contralateral side the vessels are larger than normal. In pulmonary emphysema, however, the appearance is quite striking in that the larger (proximal) vessels are abnormally dilated, while the peripheral vessels are decreased both in number and caliber.

Bronchospirometric studies confirm the fact that oxygen consumption in the lung is increased with increased vascularity and decreased with decreased vascularity, with an accompanying decrease in the caliber of the vessels and the rate of flow. Thus, with pneumography it is possible to determine the functional capacity of a diseased lung and the extent to which the normal lung is able to compensate. This information is particularly useful when surgical intervention is being considered.—*William H. Shehadi, M.D.*

Starer, F., and Sutton, David. Aortic occlusion (Leriche's syndrome) in mitral stenosis; report of six cases. *Brit. M. J.*, Aug., 1960, 2, 644–647. (From: Radiological Department, St. Mary's Hospital, London, England.)

Peripheral emboli are a frequent hazard to the patient with mitral stenosis and auricular fibrillation. The insidious onset of aortic thrombosis in patients with mitral stenosis has not been previously described. The authors report 6 cases of gradual aortic occlusion in patients with mitral stenosis and auricular fibrillation. In fact, 10 per cent of their cases with aortic thrombosis occurred in patients with mitral stenosis. In all of the authors' cases, repeated peripheral emboli preceded aortic thrombosis.

Two theories of the pathogenesis of aortic thrombosis in this group of patients are given. The first is that an extensive thrombosis involving the aorta was built up by the accumulation of repeated small emboli, most of which were too small to produce major symptoms by themselves. The second possibility is that there was progressive clotting in the vessel proximal to the peripheral embolic occlusion. On present evidence it is not possible to be certain which of the views discussed is correct.—*Eugene C. Klatte, M.D.*

Arcilla, Rene A., and Gasul, Benjamin M. Congenital dextrocardia; clinical, angiocardiographic, and autopsy studies on 50 patients. *J. Pediat.*, Jan., 1961, *58*, 39–58. (From: The Pediatric Cardiophysiology Department, Cook County Children's Hospital and Hektoen Institute for Medical Research, Chicago, Ill.)

Dextrocardia is associated sufficiently often with cardiac anomalies amenable to palliative or corrective surgery to inspire more than academic interest in the subject. The cases previously described as dextrocardia without situs inversus, and now classified as dextroversion complex or mixed dextrocardia, are almost always accompanied by severe congenital malformation of the heart. Dextrocardia secondary to anomalies of the lungs, diaphragm or chest (ex-

trinsic dextrocardia) usually has no associated cardiac abnormality.

Data on 55 cases of congenital dextrocardia were reviewed. Five were excluded because of incomplete records. (Cases of extrinsic dextrocardia were not considered.) There were 26 males and 24 females, and the ages ranged from birth to twelve years with 1 exception. Each of these patients was followed to surgery and, in 40 per cent, to autopsy.

The degree of dextrocardia was determined by the angle formed by the longitudinal axis of the heart with the midsagittal axis of the chest in the horizontal plane. This is normally 40° to the left. An angle of 30 to 40° to the right was considered to be complete dextrocardia and from 0 to 20°, incomplete. This reflects the degree of pivotal rotation of the heart along the horizontal plane in the extrinsic group, and the degree of cardiac heterotaxy in the intrinsic group.

The cases were classified into five categories:

Type I. Mirror-Image Dextrocardia (anatomic inversion of the chambers). Six of the 11 cases in this group had associated cardiac anomalies. This is at variance with other reported series, the majority of which record normal cardiac findings in this type of dextrocardia. The discrepancy is probably due to a combination of incorrect classification (several cases reported are clearly incomplete dextroversion or mesoversion) and differences in the age groups studied (those with serious cardiac anomalies die in infancy).

Type II. Dextroversion Complex (complete and incomplete mesoversion). Fourteen of the 15 cases in this group had associated cardiac anomalies. In complete dextroversion the auricles and ventricles, and with them the greater vessels, are reversed in position in the sagittal plane as if the heart had undergone 90° pivotal rotation about a posteriorly placed vertical axis. Mesoversion is similar but with less than 90° rotation so that the heart appears narrower than normal in the posteroanterior view and the relative position of the chambers is the same as ordinarily seen in a left anterior oblique view.

Type III. Mixed Dextrocardia. Either the atria alone may be transposed, or the ventricles alone, with or without transposition of the great vessels. The identification of either atrium by contrast roentgenography alone is not possible but, when a superior vena cava enters a venous atrium on the left side, atrial inversion is probably present. If there is no associated situs inversus, mixed dextrocardia should be suspected. The 4 cases of this type all had associated anomalies.

Type IV. Congenital Dextroposition. The position is the same as that which the heart would assume if it were shifted *en masse* to the right. Two of the 3 cases of this type had associated cardiac anomalies.

Type V. Extrinsic Dextrocardia. Only 4 of 17 cases showed associated anomalies.—*Lois Cowan Collins, M.D.*

Arcilla, Rene A., and Gasul, Benjamin M. Congenital dextrocardia; clinical, angiocardiographic, and autopsy studies on 50 patients. *J. Pediat.*, Feb., 1961, *58*, 251–262. (Address: Pediatric Cardiology, Cook County Children's Hospital, Chicago, Ill.)

The electrocardiographic studies in the 50 cases of congenital dextrocardia reported in the article abstracted immediately above are described for each of the five types of dextrocardia into which these cases were classified. No typical electrocardiographic findings were encountered in Type IV and the findings in these cases were normal. In the other cases the variations could be explained in most instances by the abnormal position and location of the cardiac chambers.

The roentgenologic findings in Types I, II, and III are similar in most respects, consisting of an elevated left diaphragm and identical cardiac configurations in the posteroanterior and oblique views which are the reverse of normal.

A right aortic arch is common in Type I and situs inversus always accompanies this type. In Type IV dextrocardia, the heart shadow is medially located in the frontal view and assumes normal configuration in the oblique views.—*Lois Cowan Collins, M.D.*

Lang, Erich K., and Sabiston, David C., Jr. Coronary arteriography in the selection of patients for surgery. *Radiology*, Jan., 1961, *76*, 32–38. (Address: The Johns Hopkins Hospital, Baltimore 5, Md.)

Coronary arteriography is primarily employed to determine the site and extent of arterial occlusion in the selection of patients for coronary endarterectomy. A series of 63 examinations has been evaluated. A so-called flushing technique was used in 40 examinations with satisfactory results in 32. Examinations utilizing the Seldinger technique (10 cases) and flushing with acetylcholine-induced arrest (5 cases) were unsatisfactory for the most part. In 8 cases, and particularly in the cinefluorographic examinations, direct injection into the ostium of the coronary artery has been performed with satisfactory results in all instances.

In the recent past, combined examinations using cinefluorographic techniques and the Schönander film changer have been performed in which the coronary ostium was first selectively engaged and injected for cinefluorography, and then the catheter tip was placed in the coronary cusp and the patient moved to the Schönander film changer for a second injection of contrast material and examination. Comparison of the examinations done by these two methods strongly supports the opinion that both cinefluorography and large detail film are necessary for the correct diagnosis. Electrocardiographic monitoring during the entire procedure is regarded as imperative.

Of the 41 patients examined by coronary arteriography in this series, localized lesions (amenable to endarterectomy) were present in 9, multiple lesions and peripheral lesions (generally unsuitable for endarterectomy) in 26, and a normal coronary artery in 6. Of the 6 patients operated upon (including an endarterectomy with removal of the obstruction), 5 showed good postoperative results and 1 died in the immediate postoperative period. The finding of normal coronary arteriograms in 6 patients with equivocal electrocardiographic tracing and negative Master's exercise test ruled out organic heart disease, thus helping to eliminate from surgical consideration a group of normal individuals who presented a clinical history of angina pectoris.—*Walter H. Jarvis, Jr., M.D.*

Rossetti, M. Reflux oesophagitis and bleeding oesophageal varices. *German Med. Monthly*, Jan., 1961, *6*, 1–3. (From: Department of Surgery, University of Basle, Basle, Switzerland.)

The simultaneous occurrence of esophageal varices and insufficiency of the cardia with gastroesophageal reflux is reported in 3 cases. Reflux esophagitis may cause or favor hemorrhage from the varices.

In Case I there were "idiopathic" varices and sliding hernia; in Case II "idiopathic" varices were associated with malformation of the cardia and gastric fundus; and in Case III varices secondary to portal vein thrombosis were combined with malformation of the cardia and fundus.

In each case hemorrhage from the varices occurred. In the first case transabdominal plication of the gastric fundus and under running of the venous plexus stopped the bleeding. In the other 2 cases it was necessary to perform an esophagogastrectomy.—*James J. McCort, M.D.*

Abdomen

Fournier, M., and Guien, Claude. Les stases duodénales. (Duodenal stasis.) *J. de radiol., d'électrol et de méd. nucléaire*, Dec., 1960, *41*, 757–768. (From: Service central de Radiologie de l'Hôpital de la Conception, Marseille, France.)

Duodenal stasis is more likely to occur in tall, slender, hyposthenic individuals. It appears usually between the duodenal cap and the knee of the duodenum. Concomitant findings are slight dilatation, alteration of the mucosal pattern, changes in contour and dyskinesia. The patient usually complains of a sensation of "weight" or heaviness and the feeling that the noon meal is still in the stomach long after he has eaten. This sensation disappears with borborygmus as the patient maneuvers into a position he knows will give him relief.

The roentgen signs are: (1) modification of the caliber of the duodenum, (the most revealing sign—in 1 extreme case the caliber was 9 cm.), with varying degrees of dilatation; and (2) modification of the mucosal pattern with flattening of the folds or almost total effacement; sometimes the folds present a polyhedral pattern.

The cause of stasis may be functional, organofunctional, organic or idiopathic. Functional causes may be disturbances of innervation or of muscular tonicity. The use of certain drugs is associated with disturbances of motor function; morphine causes gastric hyperperistalsis and duodenal stasis, while atropine relieves pyloric spasm and causes dilatation of the duodenum. Endocrine disturbances, particularly hypothyroidism, may be associated with stasis, which regresses under treatment. Vagotonia as a result of the Dragstedt operation may be a significant factor. Avitaminosis and acid-alkali imbalance are also mentioned as causes.

Organofunctional stasis may occur after gastrectomy or gastroenterostomy, and it may accompany gastric or duodenal ulcers, duodenal diverticulosis, and other abdominal diseases such as cholecystitis and pancreatitis. Organic stasis may occur with complete or partial duodenal obstruction. It may result from incomplete obstruction due to congenital atresia or an annular pancreas caused by ectopic pancreatic tissue. Stasis also occurs in a so-called mobile duodenum, or from extensive pressure by mesenteric vessels, neighboring organs (pancreas, kidney, biliary tract, colon, stomach), retroperitoneal neoplastic masses as well as intrinsic lesions of the descending duodenum (carcinoma, especially of the region of the ampulla, benign tumors) and foreign bodies.

Essential or idiopathic stasis with no demonstrable cause is a diagnosis that can only be made after ruling out all other causes.—*William H. Shehadi, M.D.*

Chocholáč, Joseph. (Prague, Czechoslovakia.) Les modifications de l'activité motrice de l'intestin grêle dues à une perturbation de l'afflux de la bile dans le duodénum. (Modifications in the motor activity of the small intestine due to a disturbance of the flow of bile into the duodenum.) *J. de radiol., d'électrol. et de méd. nucléaire*, Dec., 1960, *41*, 769–778.

Disturbances in the flow of bile into the small intestine may result from: (1) deficiency in the excretory function of the liver due to hepatic cellular degeneration (hepatitis, cirrhosis, ascending cholangitis, malignant disease) or to cholestasis, either primary or secondary to extrahepatic obstruction; and (2) obstruction of flow through the biliary ducts by calculi, inflammatory diseases of the biliary tract, and tumors of the biliary tract or of adjacent organs, especially the pancreas.

The author mentions the conflicting opinions

expressed in the literature on the effect of bile on intestinal motility. He also refers to the work of investigators who have demonstrated experimentally that insufficiency of bile in the digestive tract causes slow emptying of the stomach and slow transit through the intestines.

The author has studied the roentgen appearance of the small intestine in 132 patients with biliary tract disease. When an organic lesion of the stomach or duodenum was suspected, a duodenal intubation was performed to assure the passage of barium into the small intestine. In 72 per cent of these patients, the changes in the small intestine were: a decrease or lack of tonus, especially in the jejunum; dilatation of the lumen; continuity of the barium column; and suppression of peristaltic activity at the level of the involved segments. There was atony and over-all delayed transit through the small intestine.

After administering a barium meal to patients who presented the above findings, the author introduced 15 to 20 cc. of bile (8–10 per cent fel tauri) via a duodenal catheter. Within fifteen minutes, there was peristaltic activity and increased tonus of the intestines, with rapid progress of the intestinal contents.—*William H. Shehadi, M.D.*

Delannoy, E., and Paris, J. A propos d'un cas nouveau de schwannome isolé du rectum. (Concerning a new case of isolated schwannoma of the rectum.) *Presse méd.*, May 25, 1960, *68*, 1021–1022. (From: Clinique Chirurgicale des Adultes, Hôpital Régional Cité Hospitalière, Bloc Est, Lille, France.)

A sixty-eight year old patient, who had had constipation, rectal tenesmus and frequent mucous discharge over a two year period, suddenly experienced acute abdominal pain. At rectal examination, a tender, firm mass was found in the sacral concavity and bulging into the posterior wall of the rectum. At rectoscopy the mucosa was smooth and bright red in color, without ulceration or bleeding.

The clinical diagnosis was retrorectal abscess, since there was a concomitant hyperthermia which responded to antibiotic therapy. A similar attack of pain and fever occurred eleven months later and was followed by several recurrences.

By retroanal incision and resection of part of the sacrum, a tumor was removed from the rectal wall. It was easily freed from the mucosa and macroscopically resembled a fibrosarcoma.

Histologic examination demonstrated typical aspects of neurinoma with some cellular and nuclear atypical findings which indicated some possibility of malignant degeneration. No recurrence was present when the patient died fourteen months later from myocardial infarct.

This is the thirteenth such case recorded in the medical literature.—*Guy Albot, M.D.*

Mosca, Lidio G., de Di Rienzo, Nelida Vanetta, Scavino, Giovanni M., and Croppi, Carlos J. (Cordóba, Argentina.) La colecisto y la colangiografía por via oral con un nuevo medio de contraste. (Oral cholecystography and cholangiography with a new contrast medium.) *Radiología, Panamá*, Dec., 1960, *11*, 7–20.

A series of 100 cases was examined using a new contrast medium, acid beta-(3-dimethylamino-methylamino 2,4,6-triiodo phenyl)-propionate. It has an iodine content of 63.7 per cent. In most cases, six capsules of 500 mg. each were administered sixty minutes before roentgenography was done, at which time the gallbladder usually was opacified. For cholecysto-cholangiography, six capsules were administered twelve hours before the examination. If visualization was not obtained at this time, an additional six capsules were administered and the patient was re-examined in three hours. Occasionally, it was necessary to make additional roentgenograms at four and five hours after the administration of the last dose.

For cholangiography in cholecystectomized patients, six capsules were given the evening before and twelve capsules three hours before the examination. If the common duct was not visualized, additional roentgenograms were made five hours after the last dose had been given.

Positive cholecystograms were obtained in 69 per cent of the cases examined. In 66 per cent of the patients with positive gallbladders, cholangiograms were obtained. Of the cholecystectomized patients, 42 per cent also showed positive cholangiograms. Of 21 cases with nonvisualized gallbladders when they were examined with ordinary triiodized contrast medium, 24 per cent were positive when the new medium was used.

The advantages of this new contrast medium include perfect tolerance, no residual intestinal opacities, an increased percentage of visualized gallbladders and successful cholangiography even in cholecystectomized patients.—*Murray M. Friedman, M.D.*

Franke, W. Beitrag zur röntgenologischen Darstellung einer seltenen Anomalie der Gallenwege und zum Problem der postoperativen Cholangitis. (Contribution to the roentgenologic demonstration of a rare anomaly of the bile ducts and the problem of postoperative cholangitis.) *Chirurg*, Jan., 1961, *32*, 14–19. (From: Medizinische Klinik des St. Johannes-Hospital, Dortmund, Germany.)

A rare anomaly of the bile ducts is described. A secondary branch (ventral) of the right hepatic duct

communicated directly with the common duct, and the cystic duct connected with this abnormal branch. It was possible to demonstrate this anomaly because of a fistula between the pylorus and gallbladder due to a gallstone perforation with a marked stenosis of the distal end of the common duct. It is believed that this is the first case in which the anomaly has been demonstrated in a living patient.

The author does not share the optimism of some surgeons as to the long-term results of a choledochoduodenostomy. It seems almost unavoidable that there will be considerable reflux and stasis of duodenal secretion into the biliary ducts, which ultimately will lead to cholangitis. Careful roentgen examinations after choledochoduodenostomy would probably show abnormal biliary ducts more often than is now suspected.—*Hans W. Hefke, M.D.*

Grill, W. Indikation und Technik der intraoperativen Gallengangsrevision. (Indication and technique for the operative bile duct revision.) *Chirurg*, Jan., 1961, *32*, 1–9. (From: Chirurgische Universitätsklinik, München, Germany.)

The postcholecystectomy syndrome is present in 10–30 per cent of patients, depending on the evaluation of symptoms. The reason is either an erroneous diagnosis or incorrect evaluation of operative findings or technique.

Prevention of recurrent symptoms depends mainly on finding all the stones which may be present in the bile ducts or on recognizing an existing stenosis of the papilla. Stones in the ducts are palpable only in about one-half of the cases. Stenosis of the papilla cannot be palpated. Probing of the ducts fails, in two-thirds of the cases, to demonstrate stones which are present.

Operative manometry with cholangiography has proved to be the most reliable and least traumatic method for accurately evaluating the condition. The contrast material is used as the manometric fluid; usually three cholangiograms are made, although occasionally a series of them is necessary. In order to avoid changes in the muscle tonus in the biliary ducts, morphine and its derivatives are not used as preoperative medication.

Clinical examples of the method are given with operative cholangiograms. Over-filling of the ducts with contrast material may prevent demonstration of small stones. Air bubbles in the ducts can usually be recognized, but may be mistaken for stones. An erroneous diagnosis can be expected in about 10 per cent of the cases.

Mention is made of papillary stenosis and its diagnosis during operation. Obstruction and deformity of the duct by intrinsic and extrinsic tumors are also discussed.

There is practically no contraindication to this method for evaluation of the bile duct.—*Hans W. Hefke, M.D.*

Albot, G., and Delaviere, Ph. La cholostase mineure et ses variétés (cholostase mineure compensée ou décompensée avec ou sans hépatite satellite, cholostase mineure cirrhogène, cirrhose cholostatique anictérique). (Minor cholestasis and its variants [compensated or decompensated minor cholestasis with or without satellite hepatitis, minor cirrhogenous cholestasis, anicteric cholestatic cirrhosis].) *Semaine hôp. Paris*, Dec. 20, 1960, *58*, 3209–3229.

Recent knowledge of anicteric cholestatic cirrhosis has stimulated the authors to seek the means of making an early diagnosis of minor cholestasis. They believe that the findings from the various diagnostic methods should be considered together.

The earliest, most reliable and most constant signs of minor biliary stasis are histologic (portal sclerosis and Hering's neo-canals). Biologic findings are less constant (hypercholesterolemia, alkaline hyperphosphatasemia, anomalies of the bromsulphalein test). These latter are often slight but their relative increase, considered in conjunction with the clinical course, sometimes gives them a particular diagnostic significance.

The signs of diffuse satellite hepatitis (parenchymatous, mesenchymal or cirrhogenous) may be associated with the preceding signs and include the crossing-through tests or flocculation tests, generalized cell clarification or histologic signs of the cirrhogenous process.

All the signs are dependent on the relative obstruction; removal of the obstruction brings about their progressive disappearance, whether they be of histologic or biologic origin. On the basis of their interrelationship, it is possible to differentiate between compensated or decompensated minor cholestases, occurring singly or in association with a satellite hepatitis.—*Guy Albot, M.D.*

Genitourinary System

Adams, A. Wilfrid. (Bristol, England.) The movable kidney syndrome. A successful nephropexy: historical survey and current assessment. *Brit. J. Surg.*, Nov., 1960, *48*, 319–323.

"The dropped kidney" and its significance remains a matter of debate. Nephroptosis, *per se*, need not connote disease. It is asymptomatic in the majority of cases, but the so-called "movable kidney" may be associated with aching pain in the flank, dysuria and colicky abdominal distress.

The factors responsible for the syndrome are those related to the position of the kidney, the nature of its supporting investments and the amount of torsion or stress on the renal pedicle. Neurovascular elements in the pedicle may undergo stretching with resultant pain. Partial and intermittent ureteral occlusion,

which may occur, would tend to induce pyelectasis.

The author reviews the historic background of the "movable kidney" citing the lectures of Baillie in 1785 and Dietl's description in 1864 of catastrophic renal pain. Nephropexy enjoyed a vogue in the early twentieth century but by the 1930's the plea for conservatism and more careful patient selection was heard. More recently, investigators have sought objective evidence of the significance of stasis and the etiology of the pain.

Diagnosis remains difficult. Urography rarely shows pyelectasis even in the symptomatic patient, and then it is minimal. Some authorities allude to the value of the discovery of delayed excretion and they equate this sign with stasis and, therefore, infection. Functional impairment will be elucidated by advancing cineroentgenography and perfemoral aortography will be helpful in ruling out other causes of nephropathy.

Treatment today, even in cases of the true syndrome, is for the vast majority conservative with a supporting belt and rest being prescribed. Surgery is beneficial in *carefully selected* cases. The author endorses this approach and submits a case report describing a surgical success.—*Jack Reynolds, M.D.*

Spjut, Harlan J., and Nicolai, Charles H. The nonvisualizing kidney: a pathologic study of 83 nephrectomy specimens. *J. Urol.*, Jan., 1961, *85*, 115–118. (Address: Surgical Pathology, Barnes Hospital, St. Louis 10, Mo.)

A nonvisualized kidney, according to the arbitrary classification, for the purposes of this paper is defined as one in which there is no visualization of the radiopaque material in the excretory passages of the kidney on roentgenograms made over a period of time up to eight hours. If intravenous urography is not done, the kidney which does not excrete indigo carmine over a period of twenty minutes during cystoscopic examination is considered as being nonvisualized. This term is not interchangeable with nonfunctioning, although, for the purposes of comparison with subsequent pathologic findings, it has been used as such in this discussion.

The authors reviewed the roentgenograms of 255 patients who had had unilateral nephrectomy over a five year period and they studied all the histologic reports and sections from 83 nonvisualized kidneys. A minimum of three histologic sections and the gross description of the removed kidney were used as a crude estimate of potential renal function.

Pyelonephritis was found in 54 of the 83 cases of nonvisualized kidney; 7 instances of renal carcinoma were found; and, in the remaining 22, the lesions included dysplasia, renal artery aneurysm, infarction, arteriolonephrosclerosis, tuberculosis, trauma, and cysts.

Of 13 children under the age of fifteen years, 4 showed pyelonephritis, 5 had evidence of renal dysplasia, and 1 each evidence of Wilms' tumor, granulomatous disease, trauma, or infarction.

Of the 83 nonvisualizing kidneys, 62 had evidence of obstruction of various degrees, 26 contained renal stones, and 22 others were associated with obstructions such as those caused by carcinoma of the bladder, carcinoma of the cervix, and inflammatory or postoperative stenosis of the ureter.

In the group of adults, there were 5 instances where, from the study of the histologic and gross alterations of the removed kidney, the authors estimated there should be 75 per cent or more functional parenchyma. Twelve kidneys were estimated to have at least 50 per cent functional parenchyma and the remaining 66 kidneys had less than 25 per cent functional parenchyma. These estimates are admittedly crude but are correlated with the weight of the kidney and the appearance of the histologic sections.

It is noted that obstruction is an important basic factor in the pathogenesis of a nonvisualizing kidney. In those instances where the kidney is seen to be normal in size at the roentgen examination but fails to function, the authors suggest that one might evaluate the pyelogram as indicating that the kidney has a moderate amount of functional parenchyma.

It is significant that, although 20 per cent of the 83 nonvisualized kidneys did have an abundant amount of normal appearing renal parenchyma, most of these had an obstruction to account for the nonvisualization and no entirely normal kidney was found among the entire group of nonvisualized kidneys.—*George W. Chamberlin, M.D.*

Tille, D. Non-pathological filling defects of the renal pelvis and calyces. *German Med. Monthly*, Jan., 1961, *6*, 12–13. (From: Central Radiological Institute and the Radiotherapy Department, Municipal Hospital, Berlin-Buch, Germany.)

Three cases of filling defects found in the renal pelvis and calyces are reported. Combined renal percutaneous arteriography and intravenous urography demonstrated that these defects were due to overlying (but not aberrant) renal arterial branches. —*James J. McCort, M.D.*

Heitzman, E. Robert, and Perchik, Lawrence. Radiographic features of renal infarction; review of 13 cases. *Radiology*, Jan., 1961, *76*, 39–46. (Address: E. R. Heitzman, 820 S. Crouse Avenue, Syracuse 10, N. Y.)

Thirteen cases of renal infarction are presented by the authors who feel that this infrequently diagnosed condition has definite roentgen features which should lead to its more frequent recognition. Renal artery occlusion is usually the result of thrombosis, trauma, or embolism. Embolism is the most common cause and the usual history is that of abrupt onset of flank

pain in a patient with cardiac or vascular disease. Albuminuria is almost always present and hematuria is fairly common. Acute venous infarction often occurs in septic states and is accompanied by marked toxicity and almost always by hematuria.

The chief roentgen manifestation of acute renal artery occlusion is a nonfunctioning kidney of normal size in conjunction with a normal retrograde pyelogram. Segmental infarction may result in complete nonvisualization, although small infarcts may produce no abnormality or only local failure of calyceal filling. Aortography may then establish the diagnosis. Acute venous infarction also produces nonvisualization with enlargement of the involved kidney as a rule. An old total renal infarction is characterized by nonvisualization at intravenous pyelography, normal retrograde pyelograms except for diminished calyceal size, and small kidney contour.

The authors describe how this condition may be differentiated from other lesions presenting unilateral small kidneys. Recovery following sterile infarction secondary to arterial occlusion is the rule, although the affected kidney atrophies and diminishes in size. Acute venous renal infarction usually results in death unless surgical measures are instituted.—*John S. Alexander, M.D.*

Opit, L. J., McKenna, K. P., and Nairn, D. E. Closed renal injury. *Brit. J. Surg.*, Nov., 1960, *48*, 240–247. (From: University Department of Surgery, Queen Elizabeth Hospital, Woodville, South Australia.)

Two divergent opinions are expressed in the surgical literature concerning the management of renal trauma. The viewpoints of the conservatives and the interventionists are described, along with the belief of the latter that early evaluation by retrograde urography and prompt treatment by nephrectomy result in decreased morbidity and fewer late sequelae.

The authors, of conservative persuasion, review the diagnosis, management and course of 77 cases of closed renal injury in order to determine the frequency of early and late complications. Those conservatively managed patients whose clinical or pyelographic findings during the acute course could have prompted surgical intervention were closely studied. An estimate of the value of conservatism in the diagnostic method employed was also made. For the most part, diagnosis was based upon clinical findings related to the site, amount and duration of blood loss. Treatment was generally conducted without knowledge of the morphology of the lesion. Intravenous pyelography was done on only 24 patients and then just prior to their discharge. Retrograde studies were employed only in 3 cases. In 5 cases which were selected for surgery because of signs of intraperitoneal rupture or recurrent bleeding with persistent nonfunction, pyelography was employed in follow-up studies.

Seventy-two patients were treated without surgery. There were no deaths or serious morbidity during their hospital stay. Five were explored and three nephrectomies were performed. Of 34 patients available for follow-up studies, only 1 was unwell. All had good renal function but 5 showed abnormalities on the pyelograms. One patient developed hypertension after the injury.

The authors feel that their findings support conservative management of renal trauma. Concerning the use of diagnostic procedures, they suggest that intravenous pyelography will give satisfactory information in the majority of cases when it is necessary to assess morphologic damage and, in addition, will demonstrate the presence and function of the opposite kidney. Routine retrograde urography might, in itself, increase the complications and it is believed to be undesirable in the majority of patients. —*Jack Reynolds, M.D.*

Platt, William R. Bilateral renal artery aneurysm: a cause of hypertension. *J. Urol.*, Jan., 1961, *85*, 24–30. (From: The Washington University School of Medicine, Department of Pathology and Missouri Baptist Hospital, Department of Pathology, St. Louis, Mo.)

In this article, the author presents a single case report of bilateral renal artery aneurysm in a twenty-two year old male patient with hypertension. Intravenous urograms showed that there were punctate calcifications in the region of the lower pole of the right kidney and the middle of the left kidney. The calyceal system was poorly defined; the renal parenchymal shadows were normal in size; and some of the minor calyces were moderately dilated, particularly in the region of the calculi. The roentgen examination of the chest showed some evidence of enlargement of the left heart compatible with the hypertension.

The past history of this patient indicated evidence of certain neurologic disturbances which suggested at least the possibility that he may have had an aneurysm of a cerebral vessel.

During his hospital stay, the patient developed severe pain in the left loin associated with signs of bleeding into the left kidney. At operation, a massive left renal hemorrhage was found and a left nephrectomy was done. The patient died in the operating room immediately after surgery.

Postmortem examination showed left ventricular hypertrophy. There was a large dissecting retroperitoneal hemorrhage throughout the abdominal wall on the left side extending to the suprapubic region. There was a large aneurysmal cavity in the lower pole of the left kidney. The left renal artery showed an aneurysm $6 \times 4 \times 2$ cm. containing firmly adherent mural thrombi. The wall of this artery had focal areas of calcific and atheromatous change. The right kidney contained yellowish, friable calculi 3 to 6 mm. in diameter and the right renal artery showed an aneu-

rysm 2×3×5 cm. with a laminated reddish-gray mural thrombus.

Sections of the right and left kidney showed microscopically that there was thickening of the arteriolar walls and the lumina were slightly narrowed. The sections of the left kidney revealed an aneurysmal, thinned structure occupying the greater portion of the left lower pole of the kidney which compressed the adjacent pelvis and renal parenchyma. Microscopic study of the left and right renal artery showed that the intima was somewhat irregular and there was definite splitting of the elastic lamella with deposition of cholesterol and some destruction of the connective tissue fibers. The medial subintimal layers contained lipid-like material with associated hyaline-like plaques of fibrous tissue. The medial coat did not appear to be infiltrated by the fibrous elements such as are frequently associated with syphilitic lesions of the blood vessels. From these findings, it seems that the aneurysms are probably of arteriosclerotic origin with possibly a congenital weakness of the elastic fibers of the subintimal region and medial layer.

In the discussion of the etiology of hypertension, it is noted that this can occur with renal artery embolism, thrombosis, arteriosclerotic plaques, syphilitic arteritis, stenosis, and aneurysm formation. The hypertension in this particular individual could be explained by the narrowing of the orifices of the renal arteries which extended for a distance of 1 to 1.5 cm. It can be presumed, in this twenty-two year old male patient, that the bilateral renal artery aneurysms and the possible cerebral aneurysm were due to an underlying developmental defect upon which arteritis and arteriosclerotic phenomena were superimposed.—*George W. Chamberlin, M.D.*

Boeri, V., and Ramella, G. L'urografia endovenosa nei portatori di ipertrofia prostatica. (Intravenous urography in prostatic hypertrophy.) *Radiol. med.*, Aug., 1960, *46*, 708–728. (From: Istituto di Radiologia della Università di Genova, Italy.)

The authors review 210 cases of prostatic hypertrophy in which intravenous urography was done in the years 1957 and 1958. Their conclusions are as follows:

(1) Excretion urography in prostatism has a significance in the evaluation of renal function that may be compared to laboratory methods such as the blood urea, nitrogen, and phenolsulfonphthalein tests but with the advantage that it is not influenced by external factors or technical errors.

Although it does not replace clearance studies, it is useful and practical for the surgical evaluation of this type of patient.

(2) Intravenous urography permits an evaluation of the morphologic state of the entire urinary tract, of its function and of the reversibility of its lesions

(3) In the authors' opinion one of the principal advantages of excretion urography is that it allows one to decide on a therapeutic approach without the necessity of instrumentation, which is always dangerous in this type of patient: (a) good excretion, without evidence of alteration of the upper urinary tract, permits an unhurried surgical intervention determined by the general state of the patient; (b) poor excretion in the presence of nondilatation of the urinary tract indicates grave parenchymal insufficiency and requires a delay in surgery until such time as more precise studies may determine that the alterations are reversible; (c) poor excretion with dilatation of the upper urinary tract requires a period of bladder drainage before surgery, until the upper urinary tract returns to normal. Vesical drainage was obtained by a permanent catheter and only rarely was it necessary to perform suprapubic cystostomy; and (d) poor excretion associated with kinking of the juxtavesical portion of the ureter requires urgent prostatectomy, since it is impossible to improve renal function by means of vesical drainage. These cases are characterized by retention of the contrast medium in the upper urinary tract.—*Anthony A. Blasi, M.D.*

Waterhouse, Keith. Voiding cystourethrography: a simple technique. *J. Urol.*, Jan., 1961, *85*, 103–104. (From: State University of New York, Downstate Medical Center, College of Medicine at Brooklyn, and the Department of Surgery, Division of Urology, Kings County Hospital, Brooklyn, N. Y.)

Previous descriptions of the technical procedures used for voiding cystourethrography have included the rather elaborate techniques of some Swedish authors who make simultaneous roentgen-ray exposures in the lateral and anteroposterior projection while the patient is voiding a suspension of barium sulfate. To do this, it is necessary to have a special swing-like cradle to support the patient and the technical difficulties associated with this procedure make it somewhat unsatisfactory for routine performance.

The author has reduced the technique to its simplest elements. He makes one roentgen-ray exposure with the patient voiding while lying in the oblique position on the roentgenographic table. This is the customary bicycle-riding position in which one thigh is flexed on the abdomen and the leg is flexed on the thigh while the opposite extremity is straight. A small, soft catheter is inserted and residual urine is measured. A solution of 30 per cent renografin is introduced into the bladder until the patient complains of distention. The patient is then placed in the oblique bicycle-riding position and one roentgenogram is made when he is actively voiding. In very young children, a similar technique may be carried out and the child may be given a bottle while waiting for micturition.

Voiding urethrograms are important for a number of reasons but particularly for evidence of dilatation

of the posterior urethra in association with posterior urethral valves.—*George W. Chamberlin, M.D.*

FEIST, JOHN H., and LASSER, ELLIOTT C. Pheochromocytoma with large cystic calcification and associated sphenoid ridge malformation. *Radiology*, Jan., 1961, *76*, 21–26. (From: Presbyterian Hospital, Pittsburgh 13, Pa.)

Pheochromocytoma is not a rare neoplasm, but calcification within this tumor is quite unusual. When calcification does occur, it is in the form of flecks and plaques, although there is 1 reported case of a right adrenal tumor containing a laminated calcific deposit resembling a gallstone. It is reported that 10 to 30 per cent of these tumors can be demonstrated by plain roentgenography, 20 to 50 per cent by urography, and approximately 50 per cent by retroperitoneal pneumography. The majority of the tumors demonstrated on plain roentgenograms are manifested by the presence of a soft tissue mass. It is known that 5 to 10 per cent of patients with pheochromocytoma also have neurofibromatosis or another variant of the neuroectodermal syndromes. The occurrence of such associated findings justifies roentgen examinations of the calvarium and a careful search for evidence of sphenoidal ridge hypoplasia in all cases of pheochromocytoma.

The authors report a large pheochromocytoma in a seventy-four year old white male. Calcification in the capsule of a central necrotic cyst facilitated the preoperative roentgen identification. Roentgenographic examination of the skull showed gross asymmetry of the calvarium with elevation of the left petrous pyramid, attenuation of the left sphenoid ridge markings, and asymmetry of the orbits. The surgically removed tumor measured 8×7×6 cm. The pathologic diagnosis was pheochromocytoma of the right adrenal gland, with cystic degeneration. The patient died one day following surgery. Necropsy confirmed the roentgenologically predicted deformities of the calvarium and the resultant enlargement of the left middle cranial fossa.—*A. W. Sommer, M.D.*

SKELETAL SYSTEM

YENTIS, IRVAN. Radiological aspects of myelomatosis. *Clin. radiol.*, Jan., 1961, *12*, 1–7. (From: Department of Radiology, King Edward VII Hospital, Windsor, England.)

There are three main varieties of myelomatosis: (1) lesions involving the bone marrow, including multiple myeloma and solitary myeloma; (2) extramedullary lesions, involving (a) the upper respiratory tract (sinuses, larynx, and nasopharynx), (b) the lymph nodes, and (c) other sites; and (3) plasma cell leukemia. The roentgenologic changes in bone, which are considered in this paper, are important because most of the lesions are located here and are roentgenologically significant. However, in considering the bone changes it must be remembered that the invaded skeleton may look completely normal to the radiologist.

The punched-out clear cut, rounded translucencies which are said to be typical of myelomatosis are seen best in the skull vault. The internal erosion of the cortex may produce a characteristic sharply scalloped appearance. Mandibular deposits are not uncommon. Soft tissue extensions are so frequent that their presence is a useful indication of disease in the ribs, where they form subpleural nodes and masses. Local rib expansion and pathologic fracture are commonly seen. Occasionally, plasma cell tumor may have a trabeculate appearance and, if assumedly single, it may simulate osteoclastoma. However, giant cell tumors tend to appear in young people, in the twenty to thirty year age group, whereas plasmacytoma is rare before the age of forty. A very common form of bone change is osteoporosis, seen best in the spine where the uniform trabecular absorption and cortical thinning are often associated with vertebral collapse.

Hyperparathyroidism, secondary carcinomatosis, Cushing's disease, osteomalacia and senile osteoporosis may have to be considered in the differential diagnosis. While it is generally held that "primary" myelomatous bone lesions are entirely osteolytic, new bone formation may occur: (a) as callus following fracture; (b) following treatment (radiation therapy or urethane); and (c) as a result of amyloid deposition. The author has recently seen three examples of bone spiculation in multiple myeloma.

Commenting on the subject of "solitary" myeloma of bone, the author feels that such apparently localized lesions, whether in bone or soft tissue, are part of a generalized disease. These tumors are "benign" only in the relative sense of being the least virulent manifestation of a malignant process, with plasma cell leukemia being the most virulent form. The possibility of frank dissemination is present in every patient with a "solitary" lesion.—*Samuel G. Henderson, M.D.*

BROMLEY, J. F. Myelomatosis and the radiotherapist. *Clin. radiol.*, Jan., 1961, *12*, 8–16. (From: Radiotherapy Department, The General Hospital, Birmingham 4, England.)

Over the past twenty years, views about the incidence, diagnosis and prognosis of myelomatosis, as regards the prolongation of useful life, have changed considerably. The author, in this paper, discusses 87 patients registered with the Department of Medical Statistics of the United Birmingham Hospitals in the years 1950 to 1956, from the standpoint of the value of roentgen therapy.

The age distribution shows a maximum incidence in the sixty to seventy year group with a preponderance of females. Backache in one form or another is

the most common symptom; other bone pains are second, and various chest symptoms third. Cough, bronchitis, pleurisy-like pains, various forms of hemorrhage, a painful lump on the breast bone or some form of abdominal disorder may be present. In the diagnosis, examination of the bone marrow is by far the most reliable single test. The apparently poor diagnostic quality of roentgenograms may be a true indication of the real state of the patient, who is often extremely ill, helpless and uncooperative.

In the present state of our knowledge there is no one successful method of treating this disease. The methods of treatment used in this series, with the number of patients so treated, include: (a) no treatment, 22; (b) surgery only, 4; (c) transfusion only, 5; (d) urethane, 17; (e) stilbamidine only, 2; (f) nitrogen mustard 2; (g) cortisone, 1; (h) "cytamen," 1; (i) triethylene melamine, 1; and (j) roentgen therapy (plus one P^{32}), 32.

A report of Tennant (1945) is cited concerning the case of a patient who lived five years from the onset of his symptoms without any treatment, and Bayrd and Heck (1947) described a patient operated on for paraplegia, due to an apparently localized myeloma of the first lumbar vertebra, who subsequently lived seven and one-half years without any other treatment. Results of this kind cannot help inspiring a healthy diffidence in claiming any one form of treatment as superior.

Of the 32 patients in this series treated by roentgen therapy, 5 are alive and well at four, five, six, eight and nine years, respectively. Three of the above have, to date, only solitary lesions, all proved histologically. Two patients who are alive and symptom free showed multiple tumors from the beginning. A woman aged forty-five has received over a period of four years courses of roentgen irradiation to 10 different areas. A man aged fifty-six years continues to lead a useful life eight years after initial roentgen therapy.

The author believes that roentgen therapy offers the most effective method of relieving pain and keeping the patient ambulatory. Dosage is quite variable because the disease is one which particularly demands individual treatment planning. The solitary, or apparently solitary, tumor justifies a more radical order of dosage, even in the presence of a positive bone marrow examination. In the presence of initial severe anemia and weakness in a patient of the older age group, it is inadvisable to use a high dosage.

The two separate aims of the radiotherapist are quite clear. In the case of the solitary tumor it is to obtain healing and to prevent the disease from becoming generalized; when the disease is obviously generalized, the effort is directed to keeping the patient free from pain and encouraging him to leave his bed. The author has found that tumor doses of the order of 1,000 r over ten to fourteen days to individual lesions in bone can achieve this result as effectively as larger doses.

In this group of 87 patients the respective survival rates at two years are 4.5 per cent for no treatment, 5.9 per cent for urethane, and 58 per cent for roentgen therapy. The corresponding figures for three years are 4.5 per cent for no treatment, zero for urethane, and 38.7 per cent for roentgen therapy.—*Samuel G. Henderson, M.D.*

LEONARD, B. J. The pathology of myelomatosis. *Clin. radiol.*, Jan., 1961, *12*, 20–25. (From: Pathology Department, St. Mary's Hospital, London W.2, England.)

The finding of plasma cells in the bone marrow is an essential step in the diagnosis of myelomatosis. Other diseases which produce an increase in the plasma cells in the bone marrow include Hodgkin's disease, systemic lupus erythematosus, rheumatoid arthritis and carcinoma.

The normal plasma cell has an eccentric nucleus, a characteristic arrangement of the nuclear chromatin in small blocks and a deep basophilia of the cytoplasm due to a high concentration of ribonucleic acid. Myeloma cells are usually larger than normal plasma cells; they vary in size and have a less basophilic cytoplasm; the nucleus occupies a greater proportion of the cell; the cells are often multinucleated; and the nucleoli are large and distinct.

Myelomatosis is a neoplastic disease that predominantly involves those parts of the skeleton which contain red marrow in the adult. The skull, the vertebrae, the ribs, the sternum, the pelvis and the upper ends of the femora and humeri are the sites maximally involved.

Reticulosarcoma, Hodgkin's disease and chronic lymphatic leukemia have some features in common with myelomatosis. In multifocal reticulosarcoma destructive bone lesions are common. The absence of bone lesions in acute myeloid leukemia and in all forms of acute leukemia in the adult can probably be explained when it is remembered that the course of the disease is shorter than in children because drug induced remissions are uncommon.

It would appear that destructive bone lesions are not confined to myelomatosis, but are seen in an ever widening spectrum of blood diseases. Postmortem studies made in 38 of the 48 cases of myelomatosis show that the pathologic findings can be divided into a leukemic and a sarcomatous group. In 30 cases there was a diffuse plasma cell infiltration of the bone marrow, the spleen, the liver and the lymph nodes. The plasma cell infiltration in the liver was accentuated around the portal tracts similar to the distribution in chronic lymphatic leukemia. The three examples of plasma cell leukemia showed this pattern of infiltration. The findings support the concept that myelomatosis is a plasma cell leukemia with a prolonged aleukemic phase. The smaller group of 8 patients showed nodular infiltrations of the liver and spleen. The deposits were often large and very like those seen in reticulosarcoma.

Approximately 10 to 20 per cent of patients with myelomatosis develop paramyloidosis. Paramyloid is closely related to amyloid but has different staining properties, and its distribution in various organs is different from that seen in amyloidosis. Paramyloid in myelomatosis is seen mainly in the tongue, the gastrointestinal tract, the heart, the joints, the skin and the peripheral nerves. Amyloidosis developed in 6 of 48 patients with myelomatosis, in 3 of 40 with Hodgkin's disease, and in 2 of 30 with chronic lymphatic leukemia.

The association of amyloidosis with chronic lymphatic leukemia introduces the problem of what part the neoplastic lymphocyte plays in the formation of this abnormal protein. Can the lymphocyte as well as the plasma cell produce abnormal proteins? Myelomatosis, reticulosarcoma, Hodgkin's disease and chronic lymphatic leukemia are all closely related neoplastic diseases derived from the reticulum cell. They are all invariably fatal and, at least terminally, behave as malignant tumors. All these conditions give rise to destructive bone lesions and, with the exception of reticulosarcoma, they are associated with the formation of amyloid.—*Samuel G. Henderson, M.D.*

RADIOISOTOPES

Gottschalk, Raymond G. Radioactive sulphur in chondrosarcomata; distribution, concentration, and effects of S^{35} after intravenous administration to patients with chondrosarcoma. *J. Bone & Joint Surg.*, Oct., 1960, *42A*, 1239–1257. (From: Department of Pathology, George Washington University School of Medicine, Washington, D. C.)

Several recent papers have called attention to the fact that radioactive sulfur (S^{35}) may be incorporated in chondroitin sulfate within chondrosarcomas. The author gives the results of S^{35} tissue studies in 5 patients known to have chondrosarcomas and in 1 who had an undifferentiated sarcoma arising in muscle.

In distribution studies it is interesting to note that initial uptake of S^{35} is greater in hematopoietic bone marrow than it is in cartilage, but this concentration is transient, whereas the rate of disappearance of S^{35} from cartilage, and cartilage-derived tissues, seems relatively slow. Distribution figures also show that one chondrosarcoma, described as poorly differentiated, retained a fairly uniform concentration of the isotope over a period of eleven days between administration and autopsy.

This suggests that there may be some role for radioactive sulfur in the management of malignant tumors of cartilaginous origin, as other authors have suggested. A number of interesting radioautographs are presented.—*Frederick J. Bonte, N.D.*

MISCELLANEOUS

Bickel, William H., Childs, Donald S., and Porretta, Charles M. Postirradiation fractures of the femoral neck; emphasis on the results of treatment. *J.A.M.A.*, Jan 21, 1961, *175*, 204–212. (From: The Mayo Clinic and Mayo Foundation, Rochester, Minn.)

Postirradiation fractures of the femoral neck were studied in 19 patients. In 5 patients the fractures were bilateral so that the total experience included 24 fractures. The lesions for which pelvic irradiation had been given included carcinoma of the rectum and lymphosarcoma but the predominant lesion was carcinoma of the female genital tract. The interval between the initial dose of roentgen rays and the appearance of a pathologic fracture varied from five months to twelve years, the majority of fractures occurring within five years.

There was considerable variation in the amount of radiation administered and the time during which it was given. The dose to the neck of the femur ranged from 1,200 to 3,700 r. All patients were treated with 130 to 250 kv. equipment. Since the energy absorbed by bone at these energy levels is approximately twice that of soft tissue, the authors estimate that the dosage range to the femoral necks was approximately 2,400 to 7,400 rads.

The first sign of impending fracture frequently was pain, which in many cases was not sufficiently severe to stop activity. Gradual onset of difficulty was the rule, although in several cases the fractures were rather acute, following minimal stress. There was no history of severe trauma in any patient. Roentgenograms taken at the onset of symptoms often failed to show a fracture. In some cases the negative roentgen findings led the physician to suspect early metastasis to the pelvic or hip region.

The treatment used in the 24 fractures varied from none to reconstructive operations. Thirteen fractures had nonoperative care, and union occurred in all 13. It appears that if a program of crutch walking is instituted, union occurs in about the normal period for a fractured neck of the femur (twelve weeks) even though the fracture is recognized late and even though there is some varus deformity.

Seven fractures were fixed with either pins or triflange nails, and union occurred in all. Internal fixation was used when the fracture did not appear stable, as shown by progressive slipping or separation of the fragments. One patient with a nonunion had walked with and without crutches for seventeen months. A Whitman reconstruction with a cup was performed but the result was poor because of an infection. Prostheses were inserted in two instances of nonunion. In these cases the fractures had been present for five and six months, respectively, and the shaft was markedly displaced. Although the results were good the authors question whether a union

might have occurred if the fractures had been treated with simple fixation or a fibular bone graft. No instances of avascular necrosis of the femoral head were seen.

A postirradiation fracture may be difficult to differentiate from a pathologic fracture through a tumor. In the roentgenograms, an early postirradiation fracture first resembles a pseudofracture and later a true fracture. There is rarely any erosion such as one would expect in a metastatic lesion. In the course of this study the authors saw 28 pathologic fractures of the femoral neck due to malignancy but in no instance was the primary lesion in the genital tract.

The authors state that moderate doses of roentgen rays cause an early hyperemia. This promotes the osteoporosis seen in these hips before fracture occurs. There is some tissue death and fragmentation of the trabecular pattern with loss of structural strength and a predisposition to stress fractures. Microscopically, the vascular supply to the femoral head does not appear to be greatly damaged.

It is concluded that the majority of postirradiation fractures of the femoral neck will heal rapidly and well with simple nonweight bearing treatment. Surgical procedures may be necessary if the fracture is old or the position is poor. Avascular necrosis is rare.—*J. L. Williams, M.D.*

Hallerbach, H., and Renner, K. Struma maligna als Strahlenschädigung? Zugleich ein kasuistischer Beitrag zur Statistik der Struma maligna. (Struma maligna a radiation damage? Also case report contribution to the statistics of struma maligna.) *Strahlentherapie*, Sept., 1960, *113*, 110–120. (From: Strahlenklinik Professor Dr.h.c. R. Janker, Bonn, Germany.)

Statistical study of 37 patients admitted with a diagnosis of struma maligna showed that 9 of 18 patients observed for five years were "cured," and that, of 11 patients with histologically proved malignancy, 7 had five year "cures."

Although 4 of these 37 adult patients had received radiation therapy (for a gynecologic carcinoma, an arthrosis of the knee, a goiter, and symptoms probably due to thyroid carcinoma), in none did a cause and effect relationship seem plausible. However, reports indicate that one-third of struma maligna in children may stem from antecedent irradiation, so the authors recommend that irradiation (including administration of I^{131}) of the cervical region in children be undertaken only for pressing indications.—*Henry G. Moehring, M.D.*

Köhler, J. Herpes zoster nach Röntgenbestrahlung. (Herpes zoster following roentgen irradiation.) *Strahlentherapie*, Aug., 1960, *112*, 587–594. (From: Röntgen- und Strahlenabteilung der Krankenanstalten des Kreises Aue/Sa., Germany.)

A review of the author's 12 patients who developed herpes zoster after radiation therapy and of patients reported by others (a total of 97) failed to convince him of a cause and effect relationship between irradiation and herpes zoster. He states, deliberately and cautiously, that many factors *may* combine to activate a "latent" zoster virus and that perhaps roentgen irradiation is one of these factors.—*Henry G. Moehring, M.D.*

Börner, W., Moll, E., Schneider, P., and Stucke, K. Zur Problematik der Thorotrastschäden; klinische und radiologische Untersuchungen zum Verhalten von Thorium und seiner Zerfallsprodukte im Organismus; I. Mitteilung. (The problem of thorotrast injury; clinical and radiologic investigations on the content of thorium and its disintegration products in the body; first communication.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Sept., 1960, *93*, 287–297. (Address: K. Stucke, Chirurg. Univ.-Klinik, Würzburg, Germany.)

The report is based on examinations and observations of 8 patients who received intra-arterial thorotrast injections for arteriographic purposes fifteen to twenty-three years previously. In all of them, extensive laboratory studies of the blood, urine, liver function, and electrophoresis were performed. The results in none were definitely abnormal. In 2 patients specimens of the liver and spleen were obtained by surgery. Five of the 8 patients were asymptomatic. There was no apparent genetic damage. Of the 6 married patients, 1 was pregnant and 4 had healthy children.

For evaluation of concentration and localization of the thorotrast deposits, the roentgenographic examinations for search of abnormal densities and surface scintillation surveys were used. The investigation was done topographically. The maximum count representing 2 to 5 times of the O-effect was obtained over the liver and spleen. This is explainable by the storage of the thorotrast in the liver, spleen, and lymph nodes due to the known avidity of the reticulum cell system for thorotrast. A definite connection between the thorotrast deposits and the original site of injection could not be clearly established. In 1 patient, however, who had had injection into the left carotid artery, the liver and spleen showed low counts. This was not observed in other patients in whom carotid and vertebral angiographies were performed. The amount of injected material is not specified.

In 2 patients, the spleen was removed therapeutically because of high storage of thorotrast seen in roentgenograms and on scintillation counts. Histo-

logic examinations of liver biopsy specimens presented a picture of small, nodular, perilobular liver cirrhosis with deposition of thorium around the central vein and main distribution around the periportal fields. Fatty degeneration was marked but large segments of the liver parenchyma did not appear altered. The regenerative repair activity was not definitely present. The changes in the spleen consisted of marked deposition of thorium in the trabeculae with fibrosis.

The authors feel that removal of the spleen as the main thorium storage organ is indicated.—*Nathan Kriss, M.D.*

Krückemeyer, K., Lessmann, H. D., and Pudwitz, K. R. Nierenkarzinom als Thorotrastschaden. (Kidney carcinoma as the result of thorotrast injury.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Sept., 1960, *93*, 313–321. (Address: K. Krückemeyer, Städt. Krankenh., Karl Marx Str. 142a, Berlin-Neukölln, Germany.)

Carcinoma of the left kidney is reported in a forty-five year old patient who had retrograde pyelography with thorotrast nineteen years previously.

The initial pyelograms showed mild hydronephrosis due to a suspected stone on the left and minimal changes on the right. The late studies demonstrated accumulation of opaque thorotrast in the kidney parenchyma on both sides with preserved function on intravenous pyelograms on the right and complete lack of excretion on the left. A deformity of the lower pole on the left was present, indicative of a neoplasm. The scintillation counts over both kidneys were increased 17 to 23 times of the background.

On operation an adenocarcinoma was found in the lower pole of the left kidney which was markedly atrophic and hydronephrotic. The connection between thorium deposits and the carcinoma was assumed on the basis of distribution of the agent.

Generally, in animal experiments the thorotrast stimulates the connective tissue rather than the epithelium to produce malignant lesions resulting in sarcomatous growth.—*Nathan Kriss, M.D.*

Dihlmann, W. Zur Morphologie, Theorie und Problematik der Strahlenspätschäden im Zentralnervensystem. (The morphology, theory and problems of late radiation damage to the central nervous system.) *Strahlentherapie*, Aug., 1960, *112*, 567–586. (From: Neuropathologisches Institut der Universität, Tübingen, Germany.)

Late radiation damage in the central nervous system differs from local radiation damage eleswhere only in that, because of the unique character of the brain, the clinical course is much more malignant.

From 5,300–6,000 r tissue dose (in daily increments of 200 r) approaches the tolerance limit in the central nervous system. This tolerance varies. Associated factors may decrease the tolerance, *e.g.*, cerebral edema, systemic illnesses or drugs which alter vascular permeability or circulatory efficiency. On the other hand, increased tolerance may occur, as evidenced by reports of no late cerebral damage after 9,000 r tissue dose; no late damage four years after a tissue dose of 15,000 r; and late cerebral damage in only 6 of 71 patients who received 6,000 r or more cerebral dose.—*Henry G. Moehring, M.D.*

Hennig, K. Unerwünschte Radiojod-Inkorporationen und ihre Verhütung. (Undesired uptake of radio-iodine and its prevention.) *Strahlentherapie*, July, 1960, *112*, 462–466. (From: Strahlenklinik der Medizinischen Akademie, Dresden, Germany.)

Weekly direct contact measurements of radioactivity over the thyroid gland of 3 isotope technicians showed up to 1,849 counts per minute. Similar measurements in 6 euthyroid men and 4 euthyroid women 24 hours after the oral administration of 0.12 μc of radioiodine averaged 514 counts per minute. Since the average 24 hour uptake value is 55 per cent, 935 counts per minute indicate 100 per cent uptake of the 0.12 μc; and 0.12 μc maintained in the thyroid gland yields 0.3 rem, the maximum "permissible dose." Five of the thirteen weekly counts obtained in the isotope worker charged with the measurement and administration of radioiodine therapeutic doses exceeded this "permissible" count.

Control measurements excluded ingestion *via* contaminated hands. Radioiodine was being used in ion form as a sodium iodide solution (pH 5.4–7.5). Fifteen patients referred for radioiodine diagnostic study without previous exposure to radioactive isotopes sat for a short time (15–45 minutes) at a definite distance (35 and 85 cm.) from a beaker containing a therapeutic dose (16.4–25 mc) of radioiodine. In 24 hours their thyroid glands showed direct contact counts of from 35 to 8,359 per minute. Inhalation of vaporized radioiodine seemed to be the only explanation for this uptake.

The addition of sodium bicarbonate in order to alkalinize the sodium radioiodide solution (to a pH of 7.8) suppressed the uptake by inhalation to 0–54 counts per minute. Cooling the sodium radioiodide solution suppressed the uptake only when the solution was alkaline or neutral.

The author explains the inhalation not on the basis of radioiodine being incorporated in vaporized water, but by an oxidation of the iodide ion to elementary iodine; oxygen and the dissolved carbonic acid play an essential role in this process.

Alkalinization of the sodium radioiodide solution with sodium bicarbonate prevents vaporization (and inhalation) of the radioiodine.—*Henry G. Moehring, M.D*